THE HEART BEAT

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GRAPHIC METHODS IN THE STUDY OF THE CARDIAC PATIENT

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WITH 311 ILLUSTRATIONS



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To Ann

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PREFACE

The study of a cardiac patient in ancient times was based upon data collected by simple physical means Among them inspection of the patient was considered the most important In the early part of the nineteenth century palpation percussion and auscultation were perfected and diagnosis was correlated with autopsy findings

Graphic methods were first used by Marey in 1870 during the initial phase of development of experimental methods and were soon applied to the study of cardiac patients Recording of the heart beat and of the arterial pulse were the only methods used for a long time MacKenzie first Wenckebach later made several acute and accurate studies of cardiac patients by the use of mechanical methods While Einthoven was completing his work as a pioneer of electrocardiography Sir Thomas Lewis published several studies based on the use of mechanical methods These were subsequently collected and published together with classic electrocardiographic studies in his masterful work *The Mechanism and Graphic Registration of the Heart Beat* Since 1924 no comprehensive work has been published on the subject even though the innumerable developments of the last twenty five years have multiplied the number of methods and their applications

During the last thirty years the use of graphic methods has become widespread and ever more complex No manifestation of cardiac action has escaped graphic study Tracings of cardiac motions of arterial and venous pulses of cardiac sounds and of the electrical manifestations of the heart are commonly recorded and are of great help for the diagnosis To these should be added the tracings of intrathoracic and intracardiac pressures those recording the changes of the cardiovascular silhouette at fluoroscopy and the tracings recording the shaking of the body by effect of cardiac dynamics

The present book has been written in order to correlate a great mass of data and to present them with a unified concept The material will be more easily understood because all tracings presented here were obtained with similar and modern technics Data obtained by older researchers and with older technics are evaluated and briefly presented but will not be considered in detail

Evaluation of the results which can be obtained by graphic methods can be done only by correlating the data with those of cardiovascular physiology For

this reason a concise description of cardiovascular dynamics and a brief study of electrophysiology are presented in the introductory part of the book

It is possible that physicians who have specialized in a specific branch of cardiography may object to its inclusion in this book For example electro cardiologists may consider their field as separated from the others In the same way, specialists of electrokymography may consider that their method belongs to the roentgenographic disciplines and should not be included in this book while specialists of catheterization of the heart might find that the description of their method is too brief The author hopes that even a cursory glance at the correlation between the data of the various methods will explain why none of them could be omitted and why they are all interdependent

The last part of this manual was written in an attempt to correlate the data of the various methods with the clinical pictures in the various diseases con sidered as etiological entities This will make it easier for a physician to consult the book whenever a problem arises in one of their clinical cases

An appendix describing several types of apparatus has been placed at the end of the book

The index of this book has received special attention because the author feels that in any technical book consultation should be easy and rapid

The author wishes to acknowledge the help of his staff including O M Haring M D , S Contro, M D , and G Magri M D as well as A R Gold farb Ph D consulting radiobiologist for their help in the study of newer technics and E B Shafer technical secretary for the preparation of the manuscript

The experience gained through past collaboration with F G Fleischner M D and M B Rappaport E E , has been invaluable in the discussion of several graphic methods

Confirmation of diagnosis was obtained by cardiac catheterization and angiocardiography in cases of congenital heart disease of Cook County Hos pital Cardiac Center directed by Dr B Gasul

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A A L

Chicago

PART I

Introduction

CHAPTER 1

Historical Outline

The study of a cardiac patient was based a century ago upon data supplied by history and physical examination The latter used the various physical senses (especially sight hearing and touch) and consisted of the well known tetralogy inspection palpation percussion and auscultation

The study of a cardiac patient in our time is based upon the data supplied by *history physical examination* and *technical examination* It is a paradox of our specialized era that in the minds of many the technical study has become more and more dissociated from the other two branches of examination Actually the data that a specialist should collect and analyze assume a special value and lead to a correct diagnosis only if correlated with the history and the physical findings It is true that certain branches of cardiology like electrocardiography and roentgenology give certain data which can be reported without correlation Still a complete evaluation and an accurate study can be made only if one has a clear concept of the clinical problem Thus three possibilities arise

1 The practitioner reports briefly the clinical data to the specialist and the latter formulates a diagnosis
2 The specialist takes over the job of the practitioner and studies clinically the patient before making a diagnosis
3 Having no time for this job and well aware of his responsibility the specialist gives a brief report which is less exhaustive and informative than it would be possible otherwise

It is interesting to note that certain technical methods represent an extension and completion of our senses It can be said that *fluoroscopy* is a deeper and more perfect inspection *orthodiography* a more perfect percussion, *cardiography* and *sphygmography* a more perfect palpation and *phonocardiography* a more perfect auscultation On the other hand *electrocardiography* *electrokymography* and *ballistocardiography* have no counterpart in the *methods of physical examination and supply essential data which otherwise* would be unavailable, even in a rudimentary form

The graphic methods are based upon the transcription of objective manifestations of the activity of certain organs in cardiology upon the activity of the heart and vessels Over a century ago Marey[4] wrote Science meets with two obstacles the deficiency of our senses to discover facts and the insufficiency of our language to describe them The object of the graphic methods is to get around these two obstacles, to grasp fine details which would be otherwise unobserved and to transcribe them with a clarity superior to that of our words

Graphic methods translate most obscure phenomena into clearly visible variations and enable the observer to grasp at a glance the details of the normal or abnormal function of an organ

The graphic methods can be divided into four groups

1 Those dealing with mechanical manifestations
2 Those dealing with sound manifestations
3 Those dealing with electrical manifestations
4 Those using radiation methods in the study of the cardiovascular function

MECHANICAL TRACINGS

The first tracings *of the radial pulse were recorded by Marey*[4] *The fruitful* association between Marey and Potain then led to the study of the cardiogram the sphygmogram and the phlebogram (Figs 1 and 2) The names of Potain

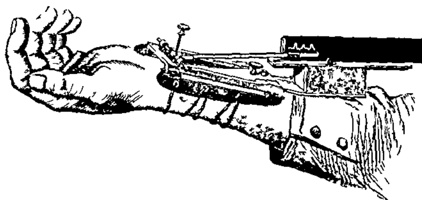


Fig 1 The first sphygmograph (from Marey)

Friedreich François Frank Dudgeon Jaquet Mougeot and MacKenzie are connected with these studies and led to the establishment of a well defined discipline In our century the names of Wenckebach Rothberger and Sir Thomas Lewis can be added to those of the older authors

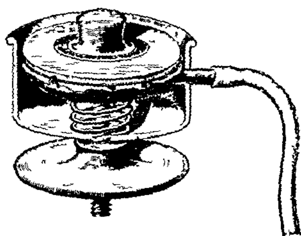


Fig 2 The first cardiograph (from Marey)

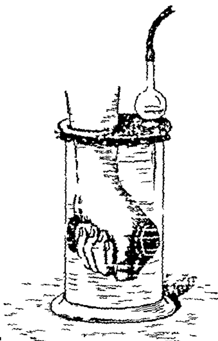


Fig 3 The first plethysmograph (from Marey)

For about twenty years the progress of electrocardiography led to a nearly complete abandonment of these methods However interest in them has been revived later by the need to use mechanical tracings as timers for the waves of the sound tracing the electrokymogram or the ballistocardiogram

Ballistocardiography even if described a long time ago has been developed only in the last two decades and is now ready to join the other branches of graphic methods in the study of cardiac patients

SOUND TRACINGS

Tracings of the cardiac sounds were recorded as far back as 1893 by Huerthle[3] Since then two methods have competed the electrical method advocated by Einthoven (Fig 4) Trendelenburg and Duchosal and subsequently perfected by Lockhart Mannheimer and Rappaport and Sprague the mechanical method advocated by Frank and further developed by



FIG 4 Electrocardiogram and phonocardiogram with Einthoven s method (from Vaquez)

Gerhartz Ohm Wiggers and Dean and Braun Menendez and Orias In the view of the author, the former technic, which now makes use of the most refined tools of electronics has become far superior to the latter

ELECTRICAL TRACINGS

Action currents were described in the animal heart by Matteucci in 1843 and were further studied by Engelmann in 1877 Ten years later Waller[8] recorded the first electrocardiograms in man Einthoven s invention of the string galvanometer[1] in 1903 avoided the need to correct the tracings and permitted the study of clinical cases (Fig 4) Since Einthoven, the names of students of electrocardiography have become a legion * Those of Hering Hoffmann, Wenckebach, Thomas Lewis Pardee and Wilson are among the most outstanding

RADIATION METHODS

This category includes all tracings recorded by means of rays, from the infrared to the visible rays and from these to the ultraviolet The most important tracings are those based upon graphic tracings of the motion of the heart as revealed by roentgen rays These studies made possible by Roentgen s discovery[6] were first developed by Stumpf s technic of roentgen kymography [7] Later on, the use of the phototube permitted the development

* Lepeschkin s book which does not include arrhythmias quotes about 10 000 articles on electrocardiography between 1934 and 1950

of the more wieldy and practical method of electrokymography described by Henny and Boone However, infrarays are used in the still developing technics of dielectrography and rheocardiography, visible rays are used in plethysmography of the fingers and toes through photoelectric recordings tracings of atomic discharges within the heart are used in radiocardiography, described by Prinzmetal [5]

It can be concluded that modern technics investigate mechanical pulsations whether slow (arterial pulse apex beat) or rapid (heart sounds), electrical pulsations (electrocardiogram) and light pulsations whether slow (visible rays) or rapid (x ray atomic discharges) Modern technical development has permitted us to transform these phenomena into electrical pulsations so that all these tracings can be simultaneously recorded on a film and compared with each other

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CHAPTER 2

The Heart Beat

The sole function of the heart is the production of a rhythmic mechanical contraction which sets the blood in motion throughout the circulatory system

The heart muscle has four basic properties automatism excitability con ductivity, and contractility A fifth tonus is under discussion

Automatism is the property of producing rhythmic stimuli It exists in all parts of the myocardium but is far better developed in the specific tissues of the heart (pacemaker and conducting system) *Excitability* is the ability to react to stimuli *Conductivity* is the ability to receive and transmit stimuli *Contractility* is the ability of the heart muscle to shorten itself thus performing work

When the heart is stimulated its reaction is of maximal intensity what ever the intensity of the stimulus In normal conditions all fibers of the myocardial syncytium have the same threshold and respond to the stimulus However variations of excitability and contractility due to either changes in the initial length of the fibers or chemical changes may modify the in tensity of reaction to a stimulus

The heart muscle does not react to a stimulus during contraction, it reacts only to a strong stimulus immediately after the end of this phase In other words the muscle is first in an absolute refractory stage then in a relative refractory stage If an abnormal stimulus causes a premature contraction the heart muscle will be in a relative refractory stage when the next stimulus

arrives, the latter will elicit no contraction and there will be the so called compensatory pause

Distention of the cardiac chambers by an increase of venous return is followed by proportionally stronger contractions (Starling s law)

In the human heart the pacemaker lies in the sinoatrial node (s a node) at the junction of the superior cava with the right atrium (Fig 5) The impulse set up by the activity of the pacemaker spreads like a wave in the atrial musculature and reaches the right atrial appendage slightly before the left Excitation of the atrioventricular node (a v node) precedes that of the right appendage Then however the progress of the stimulus meets with an important delay (higher resistance or relay of impulse?) Further progress of the stimulus takes place with greater speed in the bundle of His and its branches The endocardial surface of the septum is reached first that of the left side before that of the right However penetration from the endocardium to the epicardium is slower on the left side on account of its greater *thickness so that the left* ventricular wall is stimulated after the right

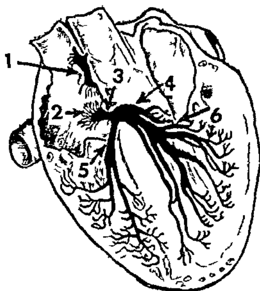


FIG 5 Scheme of the conducting system of the heart *1* S A node *2* A V node *3* bundle of His *4* right branch *5* left branch

THE ELECTROCARDIOGRAM

The human body can be considered as a medium which permits the conduction of electricity in all three dimensions Electrical currents generated in the heart reach therefore any other point of the body

In a living cell at rest the inside of the cell is negative the outside positive The cellular membrane is polarized and impermeable to electrical currents When the cells are excited a sudden decrease in the electrical resistance of the membrane takes place, the negative ions flow outwards and the difference in potential between inside and outside disappears The cell is now depolarized and its outer surface becomes negative in relation to other surrounding cells

Usually stimulation of a cell causes first a local depolarization, then a spreading wave of excitation and depolarization Following depolarization, the process of repolarization takes place resulting in a return to the previous condition

In the heart, the wave of depolarization reaches first the endocardial surface and spreads from there toward the epicardium For this reason, the balance of electrical forces is such that at any time *the endocardial surface of the heart is relatively negative in comparison to the epicardial surface* This is easily proven by cardiac catheterization and comparison of an intracardiac with a conventional electrocardiogram (Fig 6) (see p 249)

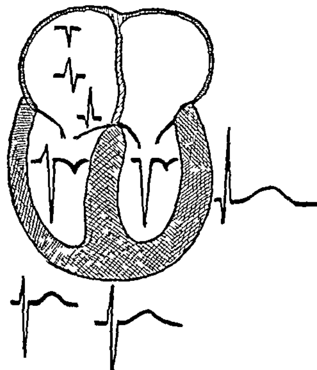


Fig 6 Intracardiac and extracardiac electrocardiogram

The electrocardiagram (ecg) is a composite curve in which various accidents or waves have a definite position These waves have been called P, Q R S and T The first part of the ecg composed of P and minor accidents is called the *atrial complex* The second part composed of QRS and T is called the *ventricular complex* (Figs 7 and 10 p 234) Details of the tracings in the various leads and their interpretation will be given in Chapters 25 28

Excitation of the s a node is revealed only by an intracardiac electrode The P wave is the result of the activation of both atria The normal Q wave represents the activation of the septum and QRS represents the period between beginning and completion of ventricular activation The T wave represents the result of ventricular inactivation

MECHANICS OF CARDIAC ACTION

Atrial Contraction

The heart cycle starts with the contraction of the atria (or atrial systole) Following the wave of excitation which spreads from the pacemaker (Fig 7) a wave of contraction starts at the openings of the cavae and spreads toward the ventricles An important backflow is prevented in normal conditions by the

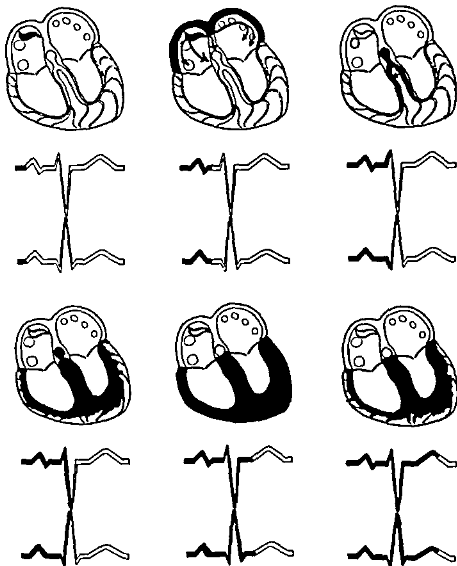


FIG 7 The various phases of excitation compared with the electrocardiogram (Ecg) Upper sketches = spreading of the stimulus Lower sketches = electrocardiogram in chest leads from the right (above) and the left side (below)

early contraction of the musculature surrounding the mouths of the large veins and by the high level reached by the venous pressure at that moment

Atrial contraction takes place in that short phase which precedes ventricular contraction and which is called *presystole* As the a v valves are already open there is only a slight rise in pressure within the atria during atrial contraction while a certain increase of ventricular pressure also takes place Atrial contraction completes ventricular filling and contributes to the normal

function of the a v valves In rapid heart action and in mitral stenosis the contraction of the atria assumes greater importance On the other hand, this contraction is absent in cases with atrial fibrillation

Ventricular Contraction

As soon as the atrial contraction is completed, the stimulus has reached the ventricles and these chambers start their contraction Each ventricle can be considered as a separate pump which maintains the flow in one direction on account of the existence of valves

During ventricular contraction all diameters of the heart decrease The contraction starts practically at the same time in both ventricles even though

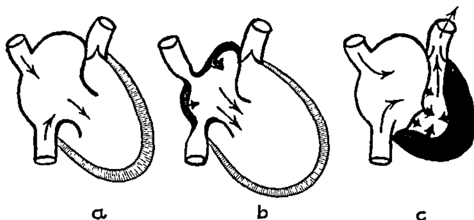


FIG 8 Atrial and ventricular contraction *a* passive ventricular filling *b* active ventricular filling (atrial contraction) *c* ventricular ejection and its effect on atrial filling

the left has a minimal delay over the right All diameters of the ventricles decrease the atrioventricular floor is pulled downwards while the apex moves but slightly upwards (Fig 8) Increased firmness of the ventricular mass and slight rotation toward the right press the apex against the chest wall and cause the so called *apex beat* or *apex thrust*

Initiation of ventricular contraction causes a rapid rise of pressure within the ventricles and closes the atrioventricular valves For a brief moment, the leaflets of these valves bulge into the atrial chambers causing a slight rise in pressure within the latter Then the contraction of the papillary muscles pulls the leaflets downwards and prevents an eversion of the valves

For a short time ventricular contraction builds up pressure within the chambers without causing any movement of blood, as shown by a steep rise in tracings of intraventricular pressure (Fig 9) This phase is called the *isometric contraction period* or *tension period* and lasts until the opening of the semilunar valves

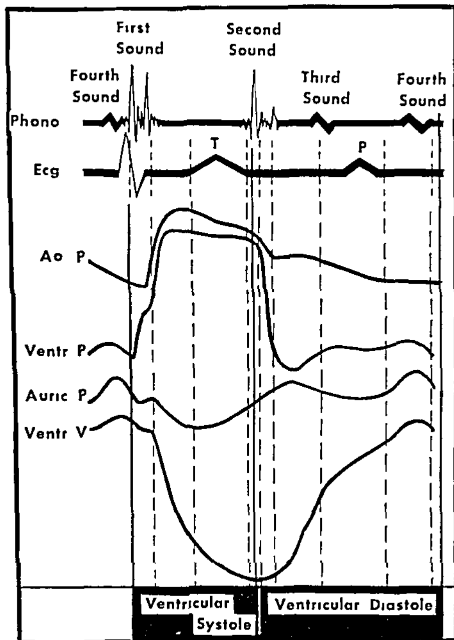


FIG 9 Atrial and ventricular pressures *Phono* phonocardiogram *Ecg* electrocardiogram *Ao P* aortic pressure *Ventr P* ventricular pressure *Auric P* atrial pressure *Ventr V* ventricular volume

As soon as ventricular pressure exceeds that of the respective artery the semilunar valves of the large arteries open and the *outflow or ejection phase* begins During the phase of ejection, the contraction of the ventricular septum and papillary muscles causes a gradual lowering of the a v floor, this leads to an increase in size of the atrial chambers and causes suction and decrease in pressure in the entire venoatrial reservoir (Figs 8 and 9) From beginning to end, ventricular pressure maintains a steady course due to the *isotonic* type of contraction This is revealed by the plateau like type of the curve of intraventricular pressure (Fig 9)

As soon as systole of the ventricles ends intraventricular pressure drops rapidly to zero The high pressure and the eddy currents of the large arteries cause closure of the semilunar valves The short interval between drop of pressure and closure of the semilunar valves is called *protodiastole* Then for a brief time, the ventricles are relaxed and empty This is the phase of *isometric relaxation* Later the pressure existing in the atria causes opening of the a v valves and filling of the ventricles begins The tricuspid valve opens about 0 05 sec after the closure of the pulmonic valve the mitral valve opens 0 06 to 0 10 after the closure of the aortic valve

Filling of the Ventricles

Filling of the ventricles takes place in three phases

1 *Rapid passive filling* in early diastole It is caused by the difference in pressure between full atria and empty ventricles The entire venoatrial reservoir experiences a drop in pressure in this phase

2 *Slow filling* during middiastole with a gradually decreasing movement of blood because the ventricles are filling up

3 *Rapid active filling* in presystole This is caused by the atrial contraction which completes ventricular filling

If there is rapid heart action the central phase is absent If the heart beats very rapidly ventricular diastole is tumultuous and takes place in only one phase If there is atrial fibrillation the third phase is absent

Table 1 summarizes the various phases of the cardiac cycle

HEART SOUNDS

Auscultation of the normal heart reveals two sounds (or tones) occasionally three Recording of the heart sounds by means of phonocardiography (p 41) often reveals four sounds (Figs 10 and 27)

The first sound takes place at the beginning of ventricular systole and lasts through the tension period and the beginning of the ejection period The second sound is shorter it takes place at the end of systole and during the phases of protodiastole and isometric relaxation

The name systolic sounds has been suggested by the author[9] for these

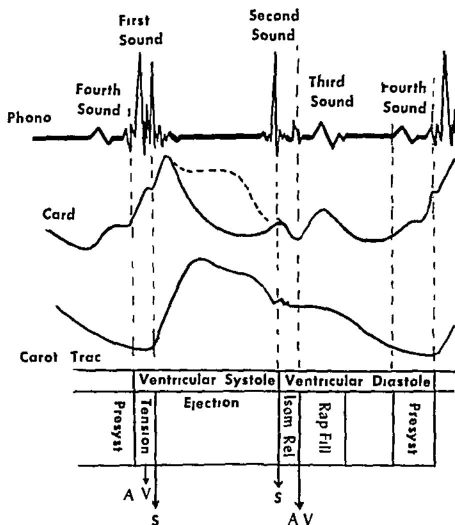


FIG 10 Vibrations of the chest wall due to cardiac action Upper tracing high frequency vibrations (phonocardiogram) Middle tracing low frequency vibrations (cardiogram) Lower tracing Carotid pulsations *A V a v valves S Semilunar valves*

two constantly heard sounds The other two less frequently heard sounds take place during diastole The name diastolic sounds has been suggested for them [9]

TIME RELATIONSHIP OF THE HEART SOUNDS

As shown by Table 1 the following dynamic phenomena take place at the time of the heart sounds

TABLE I HEART SOUNDS AND PHASES OF THE CARDIAC CYCLE

	A V Valves	Semilunar Valves	Atria	Ventricles	Phases of Cardiac Cycle	Heart Sounds	
(Presystole)			Contract	Dilate		Fourth Sound	
Systole	Close	Open	Dilate	Contract	Isotonic contraction (tension)	First Sound	Systolic Sounds
					Isometric contraction (ejection)		
		Close			Protodiastole		
	Open				Isometric relaxation	Second Sound	
Diastole				Dilate	Rapid passive filling	Third Sound	Diastolic Sounds
					Slow passive filling		
			Contract		Rapid active filling	Fourth Sound	
(Presystole)							

1 Systolic sounds

First sound	Initiation of ventricular systole Closing of the a v valves Opening of the semilunar valves
Second sound	End of ventricular systole Closing of the semilunar valves Opening of the a v valves

2 Diastolic sounds

Third sound	Rapid passive filling of the ventricles
Fourth sound	Rapid active filling of the ventricles due to atrial contraction

Mechanism of Production

First Sound Complex The mechanism of production of the complex of the first sound has been repeatedly investigated but the conclusions of the various researchers are by no means in agreement

Several authors[3 5 6 11 1] suggested a purely muscular origin of the first sound On the other hand other authors particularly Dock and Smith and coworkers[13] believe that the first sound is due to the sudden tension of the previously slack fibers of the a v valves A theory of mixed origin is advocated by Wiggers[16] who denies the possibility of separating in the sound tracing the vibrations caused by various structures and admits vibrations set up into the a v valves the chordae and the ventricular walls

The phonocardiographic studies of Orias and Braun Menendez[10] and Rappaport and Sprague[1] led to the view that the first sound is due to four separate factors (atrial muscular, valvular and vascular) However while two higher vibrations were recognized as coinciding with the two main valvular events no separate vibration or group of vibrations was found as the result of the muscular contraction

Experimental studies were conducted by the author with Alimurung and Lewis[8] It was proved that the two main vibrations of the first sound complex coincide with those two waves of the cardiogram and of intraventricular pressure tracings (p 167) which are caused by the two valvular events (closure of the a v valves and opening of the semilunar valves) It was shown that the muscular vibrations of the empty heart are extremely faint and barely appreciable It was further shown that the first sound is the result of both muscular and valvular factors Sudden changes in muscular tension activate first the a v valves and then the semilunar valves This rapid succession causes a double vibration of the cardiac wall which is further transmitted to the chest wall including high frequency (sound tracings) and low frequency (cardiogram) components Although simultaneous with the action of the

valves these vibrations are likely to arise in both the valvular and the muscular structures as a response to rapid changes in tension and pressure In other words the first sound is the audible expression of that complex movement of the heart which is also revealed by the first part of the apical thrust

SECOND SOUND COMPLEX The second sound complex is caused mainly by the closing of the semilunar valves and the resulting vibrations of the heart and the chest wall However, vibrations of vascular origin[10] and even the opening of the a v valves[7] contribute to its formation at least in certain cases

THIRD SOUND This sound arises in the ventricular wall as the result of the vibrations caused by the onrush of blood at the moment of rapid passive filling of the ventricles[4] The third sound has been attributed to valvular vibrations[14] (a theory which now is untenable and has only historical interest) or to the apical impact on the chest wall which may be only a concurrent factor, as proven by Boyer[1] It may be added that the author has recorded the third sound in a heart beating within the air chamber of a cardiometer

FOURTH SOUND This sound arises in the ventricular wall and is caused by the blood rushing into the ventricular chambers because of atrial contraction[10] Earlier vibrations can be attributed directly to the atrial contraction *per se* but can be recorded only from the esophagus

Further data on cardiac sounds will be found in Chapter 3

THE ARTERIAL PULSE

The left ventricle empties itself at each beat into the aorta This stores a portion of the blood received so that neither the pressure nor the flow fall too low before the next ventricular contraction The aorta offers little resistance to the flow of blood Its great distensibility however gives a variable resistance according to the rate at which the pressure changes

When the pressure rises suddenly (ventricular systole) the aortic volume increases considerably and this amounts to the creation of a new space (*aortic reservoir*) When the pressure falls (ventricular diastole) the retraction of the wall can be compared to the reinjection of blood from the reservoir into the aorta so that the pressure tends to be maintained in spite of the lack of flow from the heart

The pulse wave caused by the contraction of the heart travels through the aorta and large vessels much faster than the average rate at which the blood flows toward the periphery The difference disappears on the other hand, in the arterioles as the pulsating pressure is converted into steady pressure and flow

At every point of its progress, the pulse wave is accompanied by a stretching of the arterial walls resulting in the enlargement of the vessel and by an acceleration in the flow of blood At the closure of the semilunar valves the recoil of the aorta maintains the onward drive of the blood At this time the peripheral arteries are still undergoing distention but they return to their

smaller size as the excess of blood flows through the capillaries The distention caused by the emptying of the left ventricle into the aorta spreads through the arterial system toward the periphery in the form of a wave which is felt as the pulse

FUNCTIONS OF THE VEINS RESPIRATION

The return of blood through the venous system is due only partly to remaining force after it has passed through one or more capillary systems Many different mechanisms have been recognized which favor the venous return (1) contractions of the veins (2) decreasing pressure in the large veins due to the action of the heart (systolic suction) (3) aspirating effect of the low pressure existing in the thorax and increasing during inspiration (4) action of skeletal muscles on the nearest veins

Respiratory dynamics has multiple effects on the heart and on the veins During inspiration the diaphragm contracts and exerts pressure downwards As a result the following changes take place

1 The intrathoracic pressure is lowered and the intraabdominal pressure is increased favoring a flow of blood from the abdomen to the thorax
2 The liver is compressed by the diaphragm and wrung out
3 The pericardial sac is distended and its complementary sinuses open favoring diastole

As a result the blood will move from the portal circulation to the heart mainly during inspiration The blood of the lower extremities and that of the head on the contrary will show less marked changes and a more constant course Still a remarkable inspiratory collapse of the superficial veins of the neck is frequently observed

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PART II

Normal Tracings Technic and Interpretation

SECTION A

Vibrations of the Thoracic and Abdominal Walls Due to Cardiovascular Action

The movements of the heart and large vessels are accompanied by movements and vibrations of the chest wall Some of them are extremely slow and may be detected easily by inspection or bimanual palpation They include the massive movements and the see saw movement of the precordium typical of certain diseases of the heart as well as the filling up of one or more intercostal spaces during systole

Other vibrations are more rapid however, they may be appreciated by inspection or palpation They include the apex beat the multiple pulsations of the intercostal spaces and those diastolic pulsations of the cardiac apex which can be detected in cases with triple rhythms (so called gallop rhythms)

Valvular snaps like the closing snap of the aortic or pulmonic valves and the opening snap of the mitral valve are also sometimes detected by palpation

The normal heart sounds and the additional sounds which may be heard in diastole are usually detected best by auscultation even though certain diastolic sounds may escape observation on account of their low pitch

The diastolic rumble of mitral stenosis the systolic murmur of aortic or pulmonic stenosis and the systolic clicks of pericardial adhesions may be felt at palpation the former two are felt as thrills and heard as rough murmurs Last the soft murmurs of valvular insufficiency cannot be palpated and are only ausculted being of a very high pitch

CONCLUSIONS

The dynamics of the cardiovascular system causes vibrations of the chest wall which are spread over a wide frequency range from the lowest which are in the range of from 0 to 10 per second to the highest which are in the range of 600 to 1000 per second These vibrations can be detected differently by physical or technical examination according to the pitch (Table 2)

On account of the undeniable connection existing between high and low frequency vibrations of the thoracic and abdominal walls a common terminology should be used for the various waves Therefore the terms 1 2 3 and their subdivisions (1a 1b 2a 2b) which refer to the heart sounds

TABLE 2 PHYSICAL AND TECHNICAL EXAMINATION OF THE PULSATIONS OF THE PRECORDIUM

Phenomenon	*Frequency range of vibrations*	*Physical method*	*Technical method*	*Type of microphone*	*Chest piece*
General movement of chest wall See saw movement Apex beat	0–5	Inspection (palpation)	Cardiography	Linear	Large open bell
Diastolic sounds (3rd & 4th sounds gallop rhythm)	5–50	Palpation (auscultation)	Cardiography (phonocardiography)	Linear (stethoscopic)	Large open bell
Systolic sounds (1st & 2nd sounds)	10–100	Auscultation (palpation)	Phonocardiography	Stethoscopic	Large or medium open bell
Valvular snaps Diastolic rumble at apex	10–150	Auscultation (palpation)	Phonocardiography	Stethoscopic	Medium open bell
Systolic murmurs Diastolic basal murmurs Friction rubs	150–1000 (or higher)	Auscultation	Phonocardiography	Stethoscopic (logarithmic)	Open bell or diaphragm bells

should be used for the waves of the apex cardiogram (p 64) epigastric tracing (p 74), and esophagocardiogram (p 110)

The technical study of the rapid and slow vibrations of the precordium will be described in the following chapters

CHAPTER 3

Tracings of the Rapid Vibrations of the Chest

(PHONOCARDIOGRAPHY OR STETHOGRAPHY)

HISTORY

The first attempts to record heart sounds were made by Huerthle in 1893 [3] He connected a microphone with an induction coil this in turn excited a frog nerve muscle preparation which scratched a tracing on a smoked screen In 1894 Einthoven and Geluk[13] replaced the frog preparation with a capillary electrometer and obtained a graphic picture of the first heart sound Later Einthoven[14] substituted the recently invented string galvanometer to the electrometer greatly increasing the accuracy of the method (Fig 4)

During the same period a mechanical method for recording heart sounds was devised Frank[15] in 1904 experimented with his segment capsule which later became widely accepted for recording pulsations of the heart and blood vessels His method consisted of a stethoscope applied to the surface of the chest connected by a tube to a capsule covered by a rubber membrane Amplification of the vibrations was later obtained by using a light beam reflected by a small mirror with one edge cemented to the membrane The membrane was later made of living tissues [19] glass [6] celloidin [3] or gelatin [40] With this method important studies on heart sounds were made by Gerhartz [17] Garten [18] Ohm [40] and Hess [1]

vibrations ranging between 5 and 400 cycles per second while cardiac mur murs may reach 600 and only exceptionally 1000 cycles per second As a result some of the vibrations caused by the heart are inaudible and can be called *infrasounds* while most of them are only poorly audible

4 The scale of sensitivity of the ear with regard to frequency increases very rapidly so that it can be represented by means of a logarithmic ratio The *bel* is the logarithm of the ratio of two values of sound power the *decibel* is one tenth of the bel Doubling a given sound pressure increases by six decibels the sound heard whatever the original level [43]

5 In the lower frequency range the pitch may be varied rapidly without detection by the ear

6 In the presence of certain sounds the ear is unable to detect other sounds This phenomenon is known as *masking* A high pitched tone may easily mask a lower pitched tone if the frequencies are closely spaced There fore an increase in intensity obtained by instrumental means may lead to an entirely different perception, simply by revealing low pitched tones which were masked

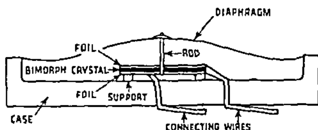


Fig 11 Cross section of a crystal microphone cartridge (from Rappaport and Sprague courtesy of the C V Mosby Co)

Microphones

A microphone is a device which transforms sound waves into equivalent elec trical pulsations The three main types in order of their development, are the carbon granule microphone the mag netic type of telephone re ceiver and the crystal micro phone Of these only the last need be considered here

The piezo electric crystal microphone (Fig 11) is based on the principle that stresses set up in a crystal of rochelle salt (or quartz) by sound waves vary proportionately the output of electric potentials The natural period of the crystal is about 10 000 cycles per second Even if this is somewhat lowered by the diaphragm it is still far above the frequency of the vibrations set up by the heart Three different adaptations of this microphone have been developed one for cardiography and two others for phonocardiography (Fig 12)

The Linear Microphone This device[39] can record all vibrations set up by cardiac action on the surface of the chest without any distortion (Fig 13)

The linear microphone satisfies Weber s criterion[50] of being *amplitudenge treu* or faithful to true amplitude

Vibrations between 0 and 1000 cycles per second are registered The value



FIG 12 The three Sanborn microphones From left to right linear stethoscopic logarithmic

RESPONSE (STETHOSCOPIC AND LINEAR)
RESPONSE (LOGARITHMIC)
180 10,000
120 1000
80 100
40 10
0 1
LINEAR
STETHOSCOPIC
LOGARITHMIC
10 000
120
100
20 30 60 100 200 400 500 1000
FREQUENCY (CYCLES PER SECOND)

FIG 13 Characteristics of the three crystal microphones revealed by the curves of amplification of the sounds

of the electrical potential is proportional to the intensity of the vibrations As the low frequency vibrations are much larger than those of the cardiac sounds (even 10,000 times) the degree of amplification needed to register the former is so small that the latter will be recorded only as minute notches, or not at all

The response of this microphone is modified by the characteristics of the amplifying galvanometer which is used In the Sanborn stetho cardiette the galvanometric channel used in conjunction with this microphone had a deflection time of about 0 01 second, more than ample for cardiographic and sphygmographic purposes In the newer twin beam Sanborn this time is further reduced to about 0 005 second About 0 2 second after a stress the system returns slowly to zero (see Appendix A) This interval is more than sufficient for cardiography and sphygmography The return to the baseline should not be forgotten in the evaluation of the slow diastolic waves of the heart

THE STETHOSCOPIC MICROPHONE In this microphone[44 45] a special case contains the cartridge and an acoustic high pass filter This filter eliminates the vibrations of extremely low frequency having importance only for the mechan ical cardiogram and protects the crystal from sudden high pressures The filtering is made by a duct or channel which allows passage of all vibrations above 30 per second while it arrests or decreases the slower vibrations More over the response of the stethoscopic microphone has a rising characteristic so that the higher the pitch of the sound the better it is transmitted (Fig 12) The resulting tracing corresponds to that auditory picture which is presented to the ear by an average stethoscope It permits the study of both relatively slow and relatively high frequencies and includes many vibrations which being barely audible or not audible can be termed infrasounds

The electrical pulsations of a stethoscopic microphone are transmitted to a galvanometer having special characteristics In the Sanborn stetho-cardiette this galvanometer is able to reproduce correctly vibrations as high as 600 per second In the twin beam the period of vibration has been further im proved so that the galvanometer of the sound tracing is able to reproduce correctly vibrations as high as 1000 per second

THE LOGARITHMIC MICROPHONE The over all frequency response of this microphone[44 4] is equivalent to the sum of the curve of the human audiogram plus that of the average acoustic stethoscope The resulting response is a graphic picture of the heart sounds as they are perceived by the average observer This is obtained by the use of a different acoustic filter attenuating the low frequency components of the sounds or murmurs As this is compen sated through greater amplification* in order to obtain a tracing of sufficient amplitude the resulting record is comparable to the clinical impression of

* The observer simply turns the knob which regulates the amplification of sounds on the panel of the stethograph The amplification increases on a logarithmic scale

the observer This is obtained by a distortion which leads to a logarithmic type of amplification of the cardiac sounds comparable to that of the human ear (Fig 12) (other deficiencies of the ear naturally cannot be introduced) The resultant tracing permits a better transcription of the high pitched or soft murmurs like the aortic diastolic murmur This microphone corresponds to the criterion of Weber[50] as being *gehoersaehnlich* or faithful to hearing because it reproduces the waves as they are heard

The galvanometer employed for recording the tracing has as already said, the ability to reproduce vibrations up to 600 per second in the stetho cardiette, up to 1000 per second in the twin beam

In summary these are the characteristics of the three microphones

Linear records practically only the slow vibrations it is used for cardiography and sphygmography

Stethoscopic records all sound vibrations, however, the very slow are filtered out the very high may be poorly transcribed if they are small it is used for a basic study of the patient

Logarithmic records all the sound vibrations distorting them in a logarith mic way as does the human ear, it is used for checking clinical impressions and for recording high pitched murmurs usually made of small vibrations even if they seem loud' upon auscultation

Amplification Systems

The latest Sanborn apparatus, the *twin beam* and the *poly beam*, have obviated the need of switching microphones during the study of a patient A single microphone is used and either a stethoscopic or a logarithmic record can be obtained by moving a switch which changes the electrical character istics of the system The linear microphone is still necessary for cardiograms or sphygmograms

A special modification of the twin beam used by the author permits the study of the transmission of murmurs through the use of two microphones of similar characteristics placed over two areas of the chest Two simultaneous phonocardiograms are recorded through separate channels It can be used also for the *simultaneous recording of two phonocardiograms one stethoscopic and the other logarithmic from the same area and with the same microphone*

It is superfluous to say that direct writing electrocardiographs cannot be used for an accurate study of cardiac sounds and murmurs Still they can be used if the heart sounds are recorded only for timing the waves of other tracings and not for the study of the fine details of sounds and murmurs

The device of Dunn and Rahm[12] is based on a single microphone The output of this is carried to three separate amplifiers having different charac teristics

1 One responds uniformly to all frequencies from 1 to 1000 per second
2 One has the lower frequencies attenuated by electrical filters
3 One has the lower and middle frequencies attenuated by electrical filters

The three tracings are recorded separately by means of cathode rays oscillographs It is apparent that the three records are basically similar to those obtained by the three Sanborn microphones However, they do not guarantee accurate stethoscopic and logarithmic types of tracing

In the device used by Mannheimer [37] a single microphone is used but six different filters lead to six galvanometers and give tracings corresponding to the following frequency ranges

(a) Below 100, (b) 50 to 175 (c) 100 to 250,
(d) 175 to 400, (e) 250 to 500 (f) 500 to 1000

Apart from the fact that the entire apparatus is unwieldy and expensive, it can be noted that several of the bands overlap, so that a correct appreciation of the actual frequency range of a sound is not easy Moreover, no over all picture is given by any of the tracings and none is comparable with clinical auscultation

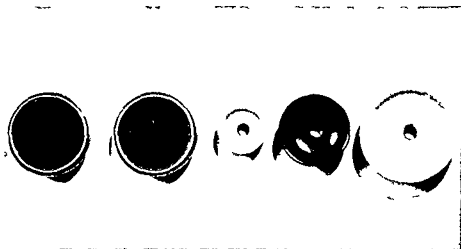


FIG 14 Various chest pieces for phonocardiography

The Chest Pieces

The choice of an adequate chest piece is important in phonocardiography A first choice will be made in regard to size (Fig 14) In the experience of the author most of the tracings should be recorded by using an open bell The largest bell able to seat without gaps over the skin of the chest should be chosen A medium sized bell will be used in adult individuals with sharply curved or prominent ribs It will also be used in young adolescents and children because a large bell picks up different types of vibrations from various points of the heart In young children, a small bell will be used It

should be kept in mind that the larger the open bell the more efficient it is in regard to low frequencies[43] However, it may present a certain resonance

A diaphragm bell is useful only in bringing out certain high pitched murmurs[43] This is obtained through suppression of low pitched vibrations while the high pitched murmurs are recorded through increased amplification of the recording instrument

The Sanborn Co supplies three open bells of 50 35, and 25 mm diameter and two membrane bells, one of them has a black rigid diaphragm 0 37 mm thick and should be used for recording high pitched murmurs the other has a brown rigid diaphragm 0 87 mm thick and should be used for the highest pitched murmurs

The use of the various microphones and chest pieces and the correlation between these and the physical means of observation of a patient have been summarized in Table 2 (p 24)

AREAS OF AUSCULTATION OF THE CHEST AND PLACEMENT OF THE MICROPHONE

Clinical experience has shown that normal heart sounds and certain abnormal murmurs are heard best in some definite areas of the chest The following suggestions on the placing of the microphone modify to a certain extent[30] the commonly accepted schemes (Fig 15)

Mitral Valve

Sounds and murmurs arising in the mitral valve are recorded best in the third and fourth left interspace at the apex or along the left ventricular border The murmur of mitral insufficiency is well transmitted toward the left axilla The murmur of mitral stenosis is louder if the patient is turned on his left side in order to increase the contact of the cardiac apex with the chest wall Both types of mitral murmurs are louder in expiratory apnea and the same applies to the opening snap of the mitral valve (p 326)

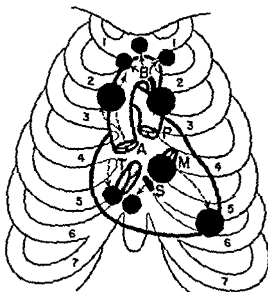


Fig 15 Areas of auscultation of the cardiac sounds and murmurs compared with position of the valves and defects *A*, aortic valve *P* pulmonic valve *T* tricuspid valve *M* mitral valve *B* ductus arteriosus *S* ventricular septal defect

The diastolic sounds of the heart (third and fourth sounds) are often louder at the beginning of inspiration or immediately after cessation of apnea They are poorly transmitted They are recorded best in the fourth and fifth interspaces or even lower and to the left in cases with left ventricular enlargement

Tricuspid Valve

Sounds and murmurs arising in the tricuspid valve are recorded best at the base of the xiphoid process or in the fourth space at the left of the sternum, in the average case at the right of the sternum, in patients with severe enlargement of the right heart Both the murmurs and the opening snap of the tricuspid valve are recorded best in inspiratory apnea [46] They are poorly transmitted

Aortic Valve

Sounds and murmurs arising in the aortic valve are usually recorded best in the second and third right interspaces and are widely transmitted However, the systolic murmur of aortic stenosis is also recorded well at the suprasternal notch [7] and along the carotid arteries The diastolic murmur of aortic insufficiency is recorded best at the left of the sternum if the heart is vertical or when the conus of the right ventricle is prominent (as in combined defects with mitral and aortic involvement) It is frequently well recorded along the left sternal border In exceptional cases it is loudest at the apex or over the xiphoid process If faint it may become louder by placing the patient in a sitting position

Pulmonic Valve

Sounds and murmurs arising in the pulmonic valve are recorded best in the second left interspace and are poorly transmitted However, the murmur of pulmonic stenosis can be recorded below the left clavicle, that of pulmonic insufficiency at the base of the xiphoid process

Ventricular Septal Defect

The murmur arising in a ventricular septal defect is recorded best in the third and fourth left interspaces near the sternum It is poorly transmitted toward the apex well toward the third and fourth right interspaces

Patent Ductus Arteriosus

The murmur arising in a patent ductus arteriosus is recorded best in the first and second left interspaces It has a wide transmission to the right of the sternum the suprasternal notch and along the carotid arteries It has wide respiratory variations

TECHNIC OF RECORDING

The first precaution to follow before recording a phonocardiographic tracing is to make sure that no superfluous extrinsic noises originate in or are carried to the phonocardiographic room They are an important cause of artifacts in the tracing The optimum conditions are found in a specially prepared soundproof room where only the patient and the operator are present during the taking of the record On the other hand fairly good tracings can be recorded in any room—even in a hospital ward—if the following precautions are observed

1 Other people in the room (students nurses other patients) should be cautioned against moving objects, walking or talking
2 Doors and windows should be closed in order to avoid as far as possible street and hospital noises No one should be allowed to enter or leave the room during the study of the patient
3 If heavy street or air traffic exists in the vicinity one should wait for occasional lulls in the noises before taking the tracing or repeat it if the noises start suddenly during registration

Respiration is a frequent cause of artifacts because the frequency of vibration of the breathing murmurs is similar to that of certain heart sounds and murmurs and cannot be filtered out electrically The construction of a special mechanical filter may be possible and is now under study in the author s laboratory

The most common artifacts (Fig 16) are caused by (a) room noises (b) respiration (c) rales (d) gastric or intestinal noises Apart from their sound characteristics all of these murmurs have no constant time relationship with the cardiac cycle and are therefore easily recognized if a sufficiently long strip is recorded

As a preliminary step the operator shall instruct the patient on how to hold his breath Most patients learn easily to stop breathing and to hold a position of intermediate apnea during each of the subsequent tracings Whenever there is difficulty the operator should close nose and mouth of the patient with his hand This maneuver is not objected to even by children if its purpose is explained beforehand It is advisable to induce sedation in infants by a phenobarbital or seconal suppository (50 mg of sedative) from thirty to forty minutes before recording the tracing

The phonocardiogram should be recorded with the patient supine or if there is orthopnea propped up by pillows over a bed or examination table

A long rubber strap is passed over the right arm and under the left armpit so that it crosses the chest obliquely The electrodes for electrocardiography are placed over the lower parts of the four limbs (p 226) and connected with the wires of the electrocardiograph The audiophone is plugged into the apparatus and placed in the ears of the observer Then a stethoscopic micro

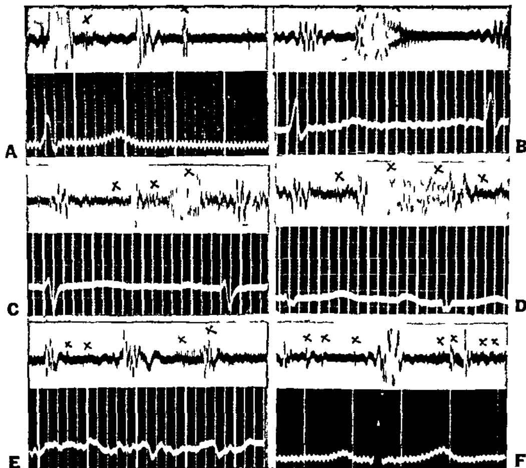


Fig 16 Artifacts in the phonocardiogram A—Extraneous noises[x] and gastric gurgle[x] B—Musical gastric gurgle[xx] C—Musical rhonchus[x] following other rhonchi[xx] D—Musicale rale[xxxx] E—Intestinal gurgle[xx] and extraneous noises[x] F—Various extraneous noises

phone is connected with the selected chest piece and placed over the chest held by the rubber strap (Fig 17)

The following areas are studied in succession in the routine examination (Fig 18) apex midprecordium pulmonic area, aortic area and tricuspid area For each of these a suitable degree of amplification is chosen one which gives an amplitude of vibrations of about 15 to 20 mm while at the same time the sounds or murmurs are heard correctly by means of the audiophone For each area the operator should record at least 5 cycles if there is bradycardia 10 cycles if there is tachycardia If the patient is unable to prolong his apnea each tracing will be taken twice in order to permit an accurate study of the various cycles

Whenever the study of a patient requires the use of special areas a special position of the patient or a special type of apnea additional records are

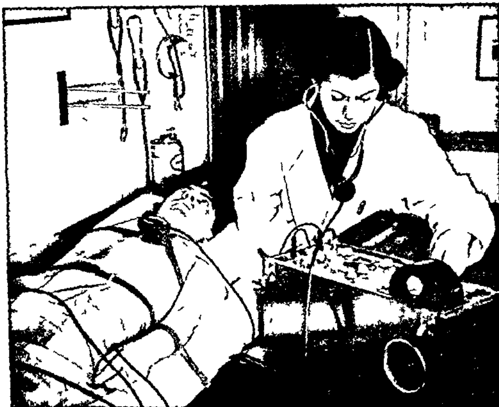


FIG 17 Technical set up for simultaneous recording of a phonocardiogram and an electrocardiogram

taken before changing the type of microphone Also, tracings may be recorded with different chest pieces Then, logarithmic tracings are recorded over the various areas and the entire procedure is repeated

If the twin beam is used first a stethoscopic and then a logarithmic phonocardiogram shall be recorded for each area The change from the one to the other is obtained simply by flipping a switch Then an increased amplification should be used

Role of Electrocardiogram

All the above tracings include the simultaneous recording of a phonocardiogram and an electrocardiogram The latter is used mostly in order to time the waves of the former Therefore the electrocardiographic lead giving the highest waves (usually lead 2 occasionally 1 or 3 or even a chest lead) will be selected All tracings are recorded with a film speed of 75 mm per second* in order to spread the sound vibrations sufficiently for study As the spreading

* Dunn and Rahm[11] advocate a speed of 200 mm per second The author believes that one of 100 mm per second would be optimal without requiring unnecessary use of film.

of the waves makes them less sharp, the voltage of the electrocardiogram may be increased to 2 or 3 cm per mv whenever the complexes are small

The electrocardiogram is helpful for the timing of three events

1 *The beginning of ventricular systole* This point has been the source of considerable discussion Practically, it can be placed at the peak of R whenever the QRS complex has a normal duration Any set of vibrations preceding R should be considered presystolic There may be a greater delay between R and the highest vibrations of the first sound in patients with pure mitral stenosis and atrial fibrillation especially when diastole is short (p 330) [8]

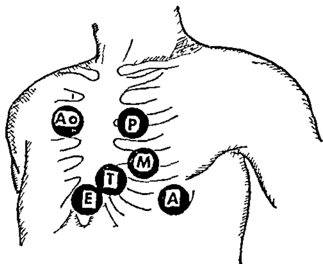


FIG 18 Areas of placement of the microphone in the routine phonocardiographic examination *A*, apex *M* midprecordium *P* pulmonic *Ao* aortic *T* tricuspid, *E* xiphoid (or ensiform) location

2 *The end of ventricular systole* Theoretically, mechanical systole should end at the end of T However, in patients with myocardial damage a dissociation between the two points is possible [20] Moreover the end of T is far from being sharp and may not be easy to appreciate if this wave is low or diphasic

3 *The interval between fourth (atrial) sound and first sound (length of P R interval)*

In addition the electrocardiogram is helpful in cases of a v block (location of atrial contractions) in patients with ectopic rhythms (recognition of the type of arrhythmia) and in cases of bundle branch block For all other purposes the electrocardiogram is a poor help and mechanical tracings should be taken as timers Therefore after completion of the first part of the examination the microphone is connected with a special chest piece (Fig 19) permitting the simultaneous recording of a linear (cardiogram) (p 64) and stethoscopic (phonocardiogram) tracing Following this the stethoscopic microphone is again placed over the most important area in the particular case and arrangements are made for recording a phonocardiogram together with a jugular tracing (p 94), a carotid tracing (p 132) or an electrokymogram (p 178)

As will be shown later (p 78) the low frequency tracing of the apex (apex

cardiogram) is the best record for timing the diastolic events of the heart in general and of the left ventricle in particular, that of the epigastrium and the phlebogram are the best in the timing of the events of the right heart the carotid tracing is useful in the timing of the events of the left heart The twin beam permits two additional studies which were not possible before

1 Comparison between a logarith mic and a stethoscopic tracing from the same area (one microphone two electrical circuits)

2 Comparison between two stetho scopic or logarithmic tracings of two different areas (two microphones two circuits) This may be useful in the study of the irradiation of mur murs and in the comparison of dif ferent extra sounds recorded over two areas

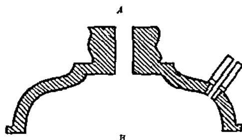


FIG 19 Scheme of the chest piece for simultaneous recording of phono cardiogram and cardiogram with two crystal microphones *A* outlet for the phonocardiogram *B* outlet for the cardiogram (From Rappaport and Sprague courtesy of the C V Mosby Co)

CALIBRATION

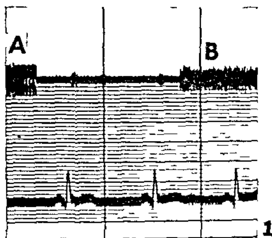
The problem of calibration of heart sounds and murmurs has been a stumbling block for several decades While a clinical tracing may be studied and discussed without reference to the absolute intensity of murmurs there is no doubt that knowledge of the latter may be of use The problem is rendered complex by the fact that several extracardiac conditions indepen dent of the recording apparatus may modify the intensity of the sounds or murmurs

A calibrator was devised by Olsen and Massa It was modified by Stodel and later by Dunn and Rahm[1] In the last device comparison was made between two juxtaposed cathode ray beams

A calibrator has been introduced in the Sanborn twin beam[52] This is based on an electrical vibration of the phonocardiographic circuit which corresponds to a period of 60 vibrations per second and a loudness of 90 decibels it is set up by pushing a button on the panel It is apparent that this device is effective only in the evaluation of the degree of electrical amplification and in the degree of weakening introduced by the various acoustic and electric filters It cannot evaluate cardiac or extracardiac causes of weakening of the vibra tions

A further study was made in the author s laboratory of the degree of inter ference brought about by the lungs and the other extracardiac tissues A small

chamber containing an electric bell is placed against the left pectoral muscle and held by a rubber strap while the stethoscopic or logarithmic microphone is placed over the area to be studied While the film is moving at a speed of 10 mm or 25 mm per second first the button of the electrical calibrator is pushed then that of the bell The sound vibrations of the latter, which have an intensity of 90 deci bels travel through the chest wall and lungs to the microphone placed at the apex or base of the heart As shown in Fig 20 the electric cali brator gives the same amplitude whether the subject is in expiration or inspiration The sound calibra tor on the other hand gives vibra tions which are louder in expira tion fainter in inspiration, varying in the same way as the heart sounds



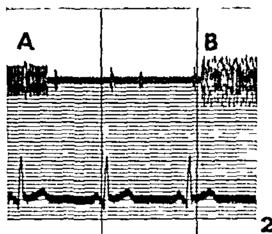


Fig 20 Calibration of heart sounds in *1* = inspiration and 2 = expiration *A*, electric calibrator *B* acoustic cali brator While the electric calibrator gives unchanged vibrations (calibration of the apparatus) the acoustic calibrator gives louder vibrations when the heart sounds are louder (calibration of the patient)

Following this procedure cali bration will be effected for each film strip and with both devices electric and acoustic with a film speed of 25 mm per second

DEVELOPING FIXING AND MOUNTING

Development and fixing of the records is made in the same way as for any bromide paper used in photography On account of the length of the film strips large tanks like those used in roentgen ray laboratories, are to be preferred *The solutions used in the above* laboratories are adequate

After drying the film will be rolled tightly for twenty four hours then unrolled and stretched over the edge of a table It may then be cut stapled to paper sheets and preserved in folders

ANALYSIS

The interpretation and analysis of a phonocardiographic record is the most important and the most difficult part of the study It is performed best by an observer who is fully aware of the clinical problem either because this has been presented to him or because he has examined the patient and has formulated the problem himself

The reading of a phonocardiogram permits only a technical diagnosis Listing of the data may be followed by the statement that these are consistent with a certain clinical diagnosis The latter will be formulated only by one in possession of both physical and technical data

The following points should be noted

1 The cardiac sounds in each cycle Relative loudness of the first and second *sound Existence and loudness of additional sounds in diastole*
2 Total duration of the first sound Duration of its various phases (pp 46 47) *Total duration of the second sound Possible splitting of this sound*
3 Existence of an opening snap
4 *Existence of a systolic murmur Time duration pitch general shape of the* vibrations Area where recorded best
5 *Existence of a diastolic murmur Time duration pitch general shape of* the vibrations Area where recorded best
6 *Existence of a presystolic murmur*
7 Recognition of friction rubs
8 *Recognition of a systolic snap*

REQUEST FOR A PHONOCARDIOGRAM PHONOCARDIOGRAPHIC REPORT

The systematic work of a laboratory of phonocardiography is made easier by the use of special slips for the request of such tracings and for reports upon them Figures 21 and 22 are examples of the slips used in the author s laboratory (pp 42 43)

SOUNDS (OR TONES) OF THE NORMAL HEART

The phonocardiogram of a normal subject may record up to four heart sounds as first shown by Houssay [2] and his pupils It is customary to call the two loudest and most commonly heard sounds (or tones) the first and the second sound As demonstrated below the first takes place during early ventricular systole the second during isometric relaxation, i e at the end of ventricular systole For this reason the author suggested that they be called *the two normal systolic sounds* [3] The other two less constant sounds are the

Name__________ Age______ Date______
Clinic
or Ward__________ Admission or Disp No ______
Referred by__________
Tentative
Diagnosis__________
Problem at
auscultation__________

	Systole	Early Diastole	Mid Diastole	Presystole
Apex				
Midpre cordium				
Pulmonary Area				
Aortic Area				
Tricuspid Area				
Extra sound or snap				

If harsh murmur mark **O**
If snap or sound, mark **M**
If soft murmur mark **V**

Fig 21 Request for phonocardiogram

third and fourth sound they take place during diastole According to the same classification they should be called *the two normal diastolic sounds*[3]

Systolic Sounds

FIRST SOUND COMPLEX This sound should be called first sound complex on account of the complexity of its configuration[30] It has been stated[6, 44] that four factors enter into its formation the atrial the muscular, the valvular, and the vascular Actually the muscular factor is revealed only indirectly through the effect that the contraction has on the position of the valves The atrial factor is to be taken into consideration only in children where the fourth sound seems to continue into the first Therefore only the vascular factor may

Name________________ Age_____ Date_____
Clinic
or Ward________________ Admission or Clinic No.________

Referred by________________

	Systolic	Early Diastolic Murmur	Mid Diastolic Murmur	Presys tolic Murmur	Second Sound	Diastolic Sound
Apex						
Midpre-cordium						
Pulmonic Area						
Aortic Area						
Tricuspid Area						

These data are consistent with a diagnosis of ________________

Fig 22 Phonocardiographic report

occasionally cause separate vibrations All other vibrations are due to a complex musculovalvular mechanism (p 17) even if separate elements or components may be recognized frequently within the sound complex

Four parts have been described within the first sound complex [6 41 44] The author prefers to recognize five

Part I is a low pitched vibration of medium height which coincides with the R wave of the electrocardiogram or slightly follows it (Fig 23) It may follow immediately a fourth (atrial) sound in young individuals but usually is well separated from it It has been considered as an atrial component of the first sound However cases with a v block or atrial fibrillation present this vibration [58] *Therefore it should be considered as coincident with and caused by* initial ventricular tension This vibration coincides with the early rise of the

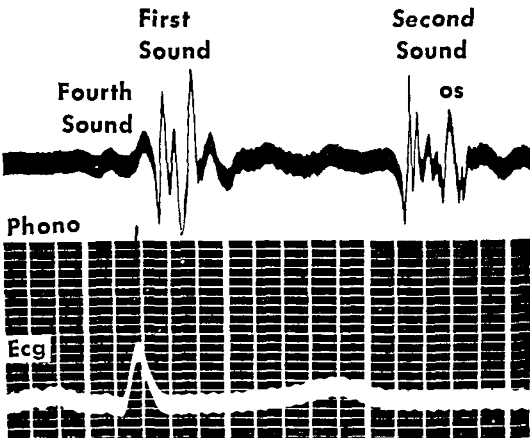


FIG 23 The heart sounds of a young adult Stethoscopic phonocardiogram and electrocardiogram Two louder vibrations are visible within the first sound complex The opening sound of the mitral valve (*os*) is clearly visible within the second sound-complex (From Luisada *et al* Brit Heart J)

apex cardiogram and falls during the descending branch of the *a* wave of the phlebogram (Fig 52) [41 44]

Part II consists of one or two large vibrations which are indirectly caused by the closure of the a v valves This part of the sound coincides with

1 A small notch in the electrokymogram of the left ventricle[54] (Fig 89A)
2 An early wave which may initiate the *c* wave of the phlebogram[30] (Fig 52)
3 The initial rise of the tracing of intraventricular pressure (Fig 32)
4 The *G* wave of the ballistocardiogram[33] (Fig 44) It precedes the rise of the pulse in the carotid and subclavian tracings

Part III is made of smaller vibrations which may include vibrations of the myocardial walls or of the chest wall It is not always visible because if its vibrations are higher they cause fusion of Part II with Part IV

Part IV consists of one or two large vibrations, similar to those of Part II

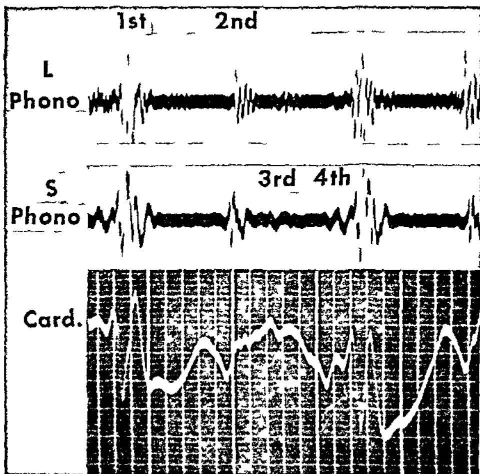


FIG 24 Logarithmic (*L*) stethoscopic (*S*) and linear (*Card*) tracings recorded at the apex in a normal child 2 years of age (superimposed)

It is caused indirectly by the opening of the semilunar valves of the aorta and pulmonary artery[3] (p 17) This part corresponds with

1 The beginning of descent of the main ventricular wave in the electrokymogram of the left ventricle (Fig 89A)
2 The rise of the pulse in the electrokymogram of the ascending aorta and pulmonary artery [54] as well as in the tracings of the suprasternal notch and of the subclavian and carotid arteries (Fig 45)
3 The second phase of the *c* wave of the phlebogram[41 44] (Fig 52)
4 The *H* wave of the ballistocardiogram[33] (Fig 44)
5 The maximum height in the tracing of intraventricular pressure (Fig 32)

Part V takes place during the ejection period [41 4] It is made of one or two

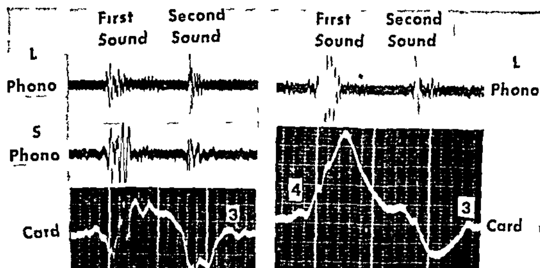


FIG 25 Logarithmic stethoscopic and linear tracings recorded at the apex in 2 normal young adults (superimposed) Symbols as in Fig 24 Waves 3 and 4 of the linear tracing correspond to the positions of the 3rd and 4th sound

slow vibrations with superimposed rapid vibrations It is caused by distention of the large arteries and by whirlpools of the blood flowing into them

Duration of the First Sound Complex The division of the first sound into five parts has important theoretical implications however a simplified method of study has practical applications, as pointed out by the author and his co workers [35] The main portion of the sound is made of large irregular vibrations (Parts II III IV) while the beginning (Part I) and the end (Part V) are made of slower vibrations It is not always easy to locate the beginning and the end of these two phases, especially the last Therefore it has been suggested[35] that the sound be divided into only three phases (Fig 29)

1 First phase small coarse initial vibrations
2 Second phase large fine central vibrations
3 Third phase small coarse final vibrations

TABLE 3 AVERAGE DURATION OF THE HEART SOUNDS THEIR PHASES AND THEIR INTERVALS

Age groups (years)	*First sound at apex (sec)* Total	1st phase	2nd phase	3rd phase	*Second sound at base (sec)* Total	1st phase	2nd phase	3rd phase	*3rd sound* (sec)	*IV I* (sec)	*II III* (sec)
Below 4	0 070	—	0 040	0 030	0 060	—	0 020	0 040	—	—	—
4–10	0 120	—	0 040	0 080	0 110	0 010	0 055	0 050	0 050	0 060	0 12
11–20	0 147	0 016	0 069	0 071	0 120	0 020	0 034	0 056	0 050	0 060	0 14
21–40	0 146	0 020	0 063	0 078	0 114	0 018	0 043	0 055	0 061	0 064	0 16
41–60	0 149	0 020	0 057	0 080	0 098	0 013	0 040	0 053	0 057	0 061	0 18
Above 60	0 141	0 024	0 050	0 080	0 085	0 010	0 038	0 044	—	0 050	—
Over all avg above age 10	0 146	0 020 (46%)	0 060	0 077	0 104	0 015 (38%)	0 039	0 052	0 059 (50%)	0 058 (78%)	0 15 (50%)

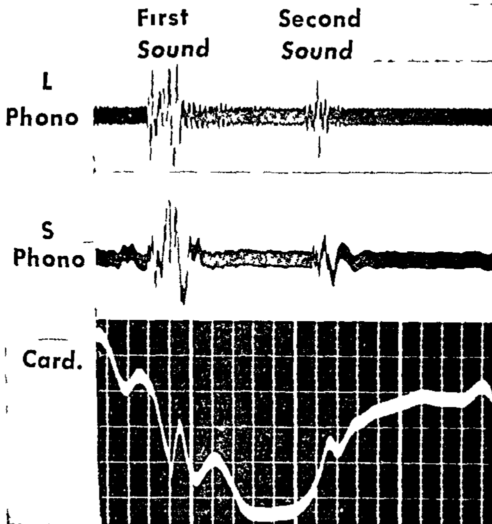


FIG. 26. Logarithmic stethoscopic and linear tracings recorded at the apex in a normal man of 70 years of age (superimposed). Symbols as in Fig. 24.

The average duration of the first sound and of its three phases can be found in Table 3 while the extreme variations are presented in Table 4.

TABLE 4. EXTREME VARIATIONS OF THE HEART SOUNDS AND THEIR MAIN PHASES

Ages	*First sound (apex)*				*Second sound (aortic area)*			
	Maximum (sec.)		*Minimum* (sec.)		*Maximum* (sec.)		*Minimum* (sec.)	
	Total duration	Second phase	Second phase	Total duration	Total duration	Second phase	Total duration	Second phase
11–20	0.16	0.12	0.12	0.04	0.12	0.04	0.08	0.03
21–40	0.22	0.10	0.09	0.02	0.16	0.10	0.08	0.03
41–60	0.22	0.10	0.07	0.03	0.14	0.06	0.06	0.02

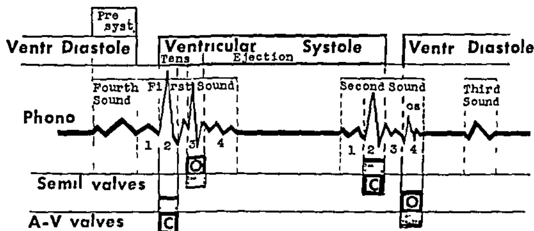


FIG 27 The valvular events of the heart and the heart sounds

A comparison of these data with those of Rappaport and Sprague[44] shows differences of the extreme figures This may be explained partly by the greater number of subjects studied by us and partly by the different method of measurement

The total average duration of the first sound at the apex with the stetho scopic microphone varies between 0 07 second in the infant and 0 149 in the age group 40 to 60 The over all average above 10 years was found to be 0 146 second

The average duration of the second phase of the first sound at the apex varies between 0 04 below age 4 and 0 069 in the age group 11 to 20 with an over all average of 0 06 second for the ages above 10 years

The maximum total duration of the first sound complex at the apex was found to be 0 16 between 11 and 20 years of age and 0 22 in the older groups The maximum duration of the second phase was found to be 0 12 in the group

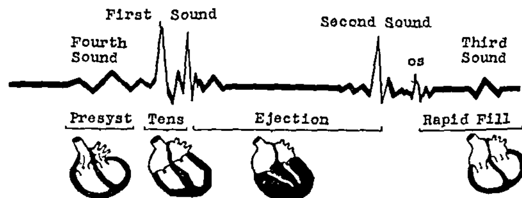


FIG 28 The heart sounds and the muscular events of the heart

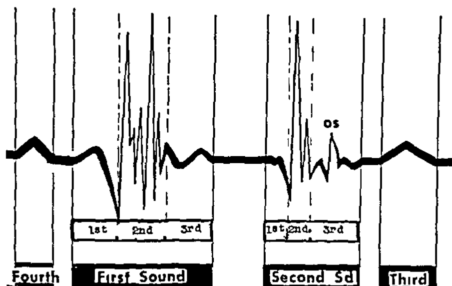


FIG 29 The three schematic phases of the first and second sounds

11 to 20 and 0 10 in the older groups Figures above these indicate the existence of a systolic murmur

SECOND SOUND-COMPLEX The second sound should be called second sound complex because it may be longer than it seems at auscultation It is caused by the rapid succession of the closure of the semilunar valves of the aorta and

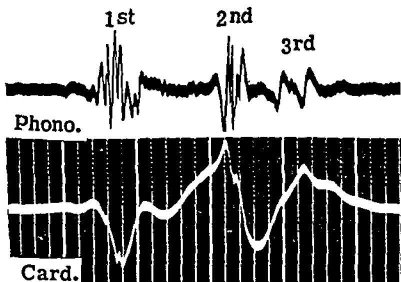


FIG 30 Multiple sounds of a normal adolescent In addition to the first and second sounds there is a split third sound

pulmonary and the subsequent opening of the a v valves (Fig 27), as well as by the causes of these valvular events (p 18) Four parts or components can be distinguished [44]

Part I consists of one or two low pitched and small vibrations caused by the beginning of isometric relaxation of the ventricles

Part II consists of one diphasic or triphasic vibration of high pitch and high amplitude It is caused by closure of the semilunar valves

This part of the sound coincides with

1 The end of the T wave of the electrocardiogram[41] (Fig 31)
2 A small notch of the apex cardiogram[30 41 44] (Figs 37, 38, and 66)
3 A small notch of the jugular tracing[41 44] (Figs 51 and 52)
4 The point *s* of the pneumocardiogram [9] (Figs 62 and 63)
5 The negative peak of the main wave of the electrokymogram of the left ventricle[34] (Fig 89)
6 The incisura of the pulse in the tracings of the carotid and subclavian tracings (Figs 38, 51 and 66) as well as in that of the suprasternal notch
7 The beginning of descent of the plateau in the tracing of intraventricular pressure (Figs 32 and 79B)

Part III consists of one or two low pitched vibrations of small amplitude resulting from the shaking of the heart and vessels and representing after vibrations

Part IV is due to the opening of the a v valves and the result of this event on the ventricular wall In most normal subjects this vibration is extremely small or even imperceptible However as proven by the author [35] normal subjects can present in this phase a single large but low pitched vibration which should be called the *opening sound* of the mitral valve (*os*) (Fig 23) Patients with lesions of the mitral or tricuspid valve may present a high pitched vibration called the *opening snap* of the mitral or the tricuspid valve (*os*)

This part of the sound coincides with

1 The lowest point of the linear tracing of the apex or midprecordium[44] (point 2b of cardiogram) (Fig 31)
2 The *v* wave of the phlebogram[30 41 44] (Fig 57) (this wave may follow *os*)
3 The point *t* of the pneumocardiogram [9] (Figs 62 and 63)
4 The point *K* of the ballistocardiogram[33] (Fig 44)
5 A notch which is often present in the electrokymogram of the left ventricle[36] (Figs 88B 89C)

Practical considerations have induced the author and his co workers[35] to divide the second sound into three phases (Fig 29)

1 First phase small inaudible vibrations
2 Second phase large central audible vibrations

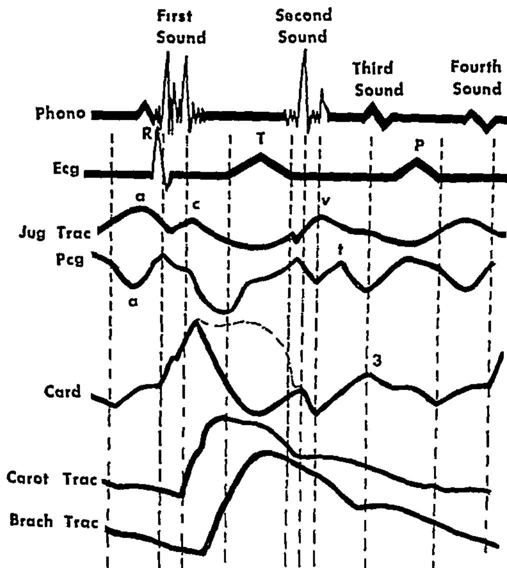


FIG 31 Comparison of the phonocardiogram with the electrocardiogram jugular tracing pneumocardiogram cardiogram carotid tracing and brachial tracing

3 Third phase small final vibrations usually but not always inaudible

Duration of Second Sound Complex The average duration of the second sound complex and of its phases can be found in Table 3 and the extreme variations in Table 4 (pp 46 and 47)

The average total duration of the second sound at the base with the stetho scopic microphone varies between 0 06 second below the age of 4 and 0 12 second in the age group 11 to 20 The average duration of the second phase

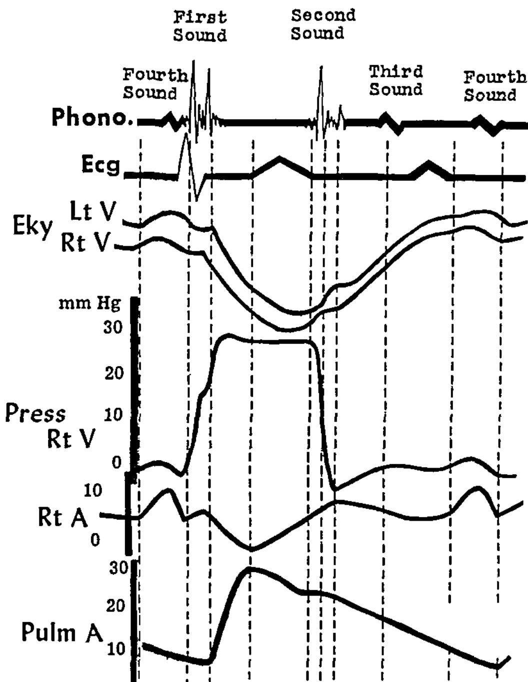


FIG 32 Comparison of the phonocardiogram with the tracings of atrial and ventricular pressure (catheterization of right heart) and with the electrokymogram (*Eky*) of the left and right ventricles

of this sound varies between 0 020 second below the age of 4 and 0 055 in the age group 11 to 20

The extreme variations are a maximum of 0 16 second for the total sound and 0 10 for the second phase, in the age group 21 to 40, a minimum of 0 06 for the total sound, and of 0 02 for the second phase in the age group 41 to 60

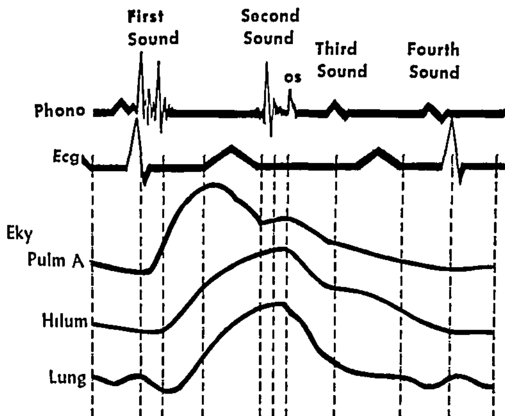


FIG 33 Comparison of the phonocardiogram with the electrokymograms of various sections of the pulmonary circulation

Diastolic Sounds

The diastolic sounds are usually two, the *third* and *fourth sounds* They may be fused in a single sound complex whenever there is severe tachycardia Calo[7, 8, 9] advocated the existence of a *fifth sound* following the fourth caused by an elastic reaction of the ventricular wall after the rapid filling This sound which appears from 0 08 to 0 16 second after the third sound has been considered by the author[31] as the result of splitting of the third sound (Fig 30)

THIRD SOUND The third sound is not always recorded and is even more

rarely audible It is more common in children adolescents, and young adults with a slender body and a flat chest It consists of a single slow vibration, usually not larger than one third or one fourth of the second sound (Figs 25, 38, 44, and 66) The third sound occurs at the peak of that early phase of diastole which has been called rapid passive filling of the ventricles (p 14)

The third sound coincides with

1 The peak of the wave *3* of the apex cardiogram[30, 44] (Figs 25, 30 and 38)
2 The point *d2* of the pneumocardiogram [29]
3 The end of rapid relaxation in the electrokymogram of the left ventricle [34, 36]

It follows the *v* wave of the phlebogram[41, 44] but may occasionally coincide with it[30] (Fig 53B) It slightly precedes the peak *L* of the ballistocardiogram[33] (Fig 48)

As shown in Table 3 the third sound has an over all average duration of 0 059 second and may be as short as 0 05 and as long as 0 061 [35]

The third sound is recorded best at the apex, especially in the left decubitus, but may be recorded well at the epigastrium (p 74) Tachycardia renders the third sound louder

FOURTH (ATRIAL) SOUND The fourth sound is seldom audible under normal conditions It may be heard occasionally in complete a v block On the other hand, this sound may influence during auscultation the character of the first sound so that the latter may be heard as booming snapping or loud when the fourth sound is louder than usual It is recorded frequently as a small low pitched vibration having a height of about one third to one fourth of the first sound (Figs 44 66) It may consist of a diphasic or even triphasic vibration, and may be followed by a small aftervibration

The fourth sound is recorded best at the apex or midprecordium especially in the left decubitus but may be recorded at the epigastrium as well (p 74) It is favored by tachycardia It is more common in children and adolescents

The fourth sound is only indirectly related to the atrial contraction A com parison between tracings recorded via the esophagus (Fig 59) and tracings recorded at the apex has shown that the fourth sound takes place earlier in the former than in the latter [41] This shows that the sound recorded at the apex is only the result of an impact of the blood pushed by the atrial contraction against the ventricular wall The fourth sound takes place at the peak of the third phase of ventricular diastole namely that of rapid active filling due to the atrial contraction (p 14)

The fourth sound has the following time relationship with the waves of other tracings

1 It follows the peak of the P wave of the electrocardiogram in Lead 2 by an interval of from 0 018 to 0 074 second (average 0 043) [41] In infants it begins at least 0 06 second after the rise of P

2 It begins after the rise of the *a* wave of the phlebogram[41] and may coincide with the peak of this wave[30, 44] (Fig 57)
3 It coincides with the peak of the *a* wave of the apex beat tracing[30, 44] (Fig 25) and of the epigastric cardiogram[30] (Fig 41)
4 It coincides with the *a* wave of the pneumocardiogram [9] (Figs 61 and 62)
5 It coincides with the point G (and occasionally the point O) of the ballistocardiogram[33] (Fig 48)
6 It coincides with a small positive, atrial wave of the electrokymogram of the left ventricular wall with the descending branch of the negative atrial wave in the eky of the right or left atrial walls[34] (Figs 90 and 91)

NORMAL INTERVALS BETWEEN THE HEART SOUNDS

The normal heart sounds are separated by intervals which do not exceed certain limits and which should be known Table 3 shows these intervals in the various age groups (p 46)

INTERVAL BETWEEN BEGINNING OF THE SECOND SOUND AND BEGINNING OF THE THIRD This may be as short as 0 12 second at the apex in children 4 to 10 years old and as long as 0 18 second at the base in persons between 41 and 60 The over all average is 0 15 second at the apex and 0 17 at the base in persons above 10 years of age

INTERVAL BETWEEN BEGINNING OF THE FOURTH SOUND AND BEGINNING OF THE FIRST The minimum is 0 05 second at the base between 41 and 60 years of age the maximum is 0 072 at the apex in persons between 21 and 40 The over all average is 0 058 upon all areas of the precordium

INTERVAL BETWEEN FIRST SOUND AND SECOND SOUND This varies according to the various lengths of ventricular systole Duration of electrical systole varies according to the heart rate (p 238) mechanical systole is slightly shorter than electrical and can be reckoned as the length of electrical systole minus one half of the duration of QRS in other words mechanical systole is about 0 04–0 05 second shorter than electrical systole

INTERVAL BETWEEN THIRD AND FOURTH SOUNDS This varies considerably according to the duration of ventricular diastole If diastole is short as in tachycardia this interval may disappear altogether

MURMURS

Systolic Murmurs

A systolic murmur is revealed by the phonocardiogram as a series of vibrations of different pitches Only in certain cases does the tracing present regular vibrations of the same frequency then auscultation reveals a musical or sea gull cry murmur

The following types of systolic murmurs can be observed (Fig 34)

1 *Prolongation of the second phase* of the first sound while the over all length of the sound is within average or maximal limits (p 46) The murmur is minimal but undeniable, as proven by Barrera Cañedo[2]

2 *Prolongation of the total duration* of the first sound which lasts beyond the peak of the c wave of the jugular tracing or even the peak of the main wave of the carotid tracing

3 *Murmur in decrescendo* (Fig 146A) This has louder vibrations at first smaller vibrations later, and prolongs the first sound It usually ends long before the second sound It is composed of vibrations of different pitches with predominance of the higher It is typical of mitral regurgitation and is recorded best at the apex in the left decubitus

4 *Diamond shaped*[6] or *pulse wave like murmur* (Fig 171B) The vibrations start soon after the end of the first sound increase toward the middle of systole decrease later They are made of various frequencies with predominance of the medium type The murmur is typical of absolute or relative stenosis of the aorta or pulmonary artery The peak of the murmur corresponds to that of the pulse in the carotid tracing The stenosis is usually not too severe and both heart sounds preserve their individuality

5 *Loud, all systolic murmur* typical of severe aortic (Fig 170) or pulmonic stenosis (Fig 189) It covers the first sound lasts through all systole, and ends with the second sound which may not be visible The vibrations are loud from the beginning and continue loud either to the middle of systole or to the end It is recorded over the second right or the second left interspace

6 *Low grade all systolic murmur* usually made of high pitched vibrations (Fig 146C) It lasts throughout all systole but does not cover the second sound It is recorded mostly at the apex and occurs in mitral regurgitation, especially in children It is a rather uncommon type of murmur

7 *Concertina like murmur* The vibrations show phases of louder and lower intensity all within a rather narrow band of frequencies The murmur has the maximum intensity over the midprecordium is rather musical and spreads in several directions It is found in myocardial infarct, following rupture of a chorda tendinea or possibly because of mural thrombi of the left ventricle It is also found in certain cases of calcific aortic stenosis, especially in older patients

8 *Crescendo systolic murmur* (rare)

Basal Diastolic Murmurs

Different types can be recognized (Fig 34)

1 *Prolongation of the second aortic or pulmonic sound* The second sound is made of three or four vibrations in decrescendo It is typical of initial aortic or pulmonic insufficiency (Fig 160)

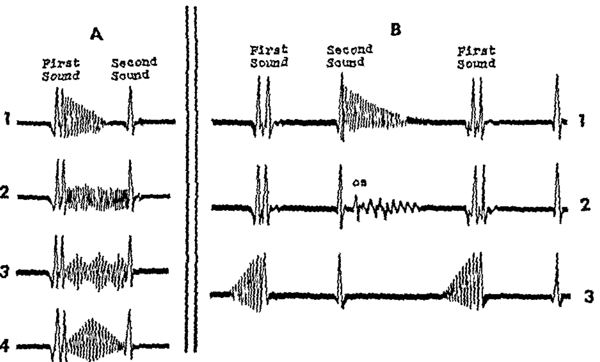


FIG 34 *A* Various types of systolic murmurs *1* apical murmur in decrescendo *2* apical continuous murmur *3* basal or apical concertina murmur *4* basal diamond shaped murmur *B* Various types of diastolic murmurs *1 2* basal diastolic murmurs in decrescendo *3* apical diastolic rumble

2 *Diastolic murmur in decrescendo* typical of advanced aortic or pulmonic insufficiency The murmur starts as a prolongation of the second sound and gradually decreases in intensity until it disappears during mid diastole If the vibrations are regular, there is a sea gull cry type of murmur The latter is more common in but not exclusive of the everted aortic valve (Fig 160)

3 *A brief interval* between the second sound and the onset of the murmur This starts in early diastole, reaches a high intensity at the time of rapid filling then gradually decreases It is not unusual in aortic insufficiency [51]

4 The diastolic murmur of aortic insufficiency has a poor intensity, it is slightly louder during the rapid filling then continues during most or all of diastole with a concertina like type There may be a further phase of increase during atrial contraction

Apical Diastolic Murmurs

Frequently the murmurs are loudest over the midprecordium and should be called rumbles on account of the predominance of low frequency vibrations (Fig 34)

1 *Early diastolic rumble* Following the second phase of the second sound, there is a pause of silence then an opening snap of the mitral valve (Fig 153) This vibration is followed immediately by a variable number of irregular vibrations at times only three or four One or two louder vibrations may correspond to the peak of the wave of rapid filling [19] On the other hand the vibrations may continue throughout most of diastole increase in presystole and continue until the following first sound (Fig 155)

2 *Presystolic murmur* (Fig 155C) It corresponds to that short phase of diastole preceding the first sound during which atrial contraction occurs It frequently is *in crescendo* and its vibrations continue with those of the first sound However in certain cases the murmur decreases before the beginning of the first sound and the auscultatory impression of a crescendo like murmur is not confirmed by the tracing (Fig 156) [1] The vibrations are usually of various frequencies but the low pitched predominate

Continuous Murmur

This type of murmur is typical of fistulas between vessels It frequently has the auditory type of a *machinery murmur* and is found in patent ductus arteriosus (Fig 196) and arteriovenous fistulas (Fig 261) The duration of the murmur is such that it does not coincide exactly with the cardiac phases it usually is loudest at the end of systole covers the second sound and then decreases in diastole There may be a concertina like murmur The frequency of the vibrations varies and several bands are usually represented as proven by the use of different filters or microphones

SPECIAL APPLICATIONS OF PHONOCARDIOGRAPHY

Fetal Phonocardiogram

The first tracing of fetal sounds was recorded by Pestalozza in 1891 [4] Since then many researchers have published fetal tracings with a gradually improving technic Recent contributions have been published by Cesa and Seganti[10] and Jordan and Randolph [24]

The method of fetal phonocardiography is relatively simple and far easier than fetal electrocardiography It permits evaluation of the rate of the fetal heart and even prenatal appreciation of severe malformations of the heart

The stethoscopic microphone with the large chest piece is applied over that part of the maternal abdomen where auscultation reveals fetal heart tones Simultaneously with the fetal sound tracing a maternal electrocardiogram is recorded Comparison of the two proves whether or not the sounds auscultated were fetal heart tones The findings will be discussed in part III (p 271)

Esophageal Phonocardiography

Extensive studies with this method have been made by Taquini[47] and later, by Miller and Groedel [38] Interest of the method lies in the fact that the heart sounds are collected from inside the chest their origin is near the collecting chamber and their transmission is not altered by bony structures Thus the fourth (atrial) sound takes place earlier than at the apex and may be due to the atrial contraction itself and not to its effect [4] The phonocardiogram is recorded by using a stomach tube closed at its end (p 110) similar to that employed for esophagocardiography The most interesting tracing is that obtained at the atrial level (Figs 58 and 59) There atrial sounds and mitral murmurs are more distinctly recorded than from the surface of the chest In general sounds and murmurs have lower frequencies than when recorded by conventional technic The heart sounds are shorter

Tracheal Phonocardiography

Tracheal phonocardiograms have been recorded by Groedel and Miller[38] in patients with a tracheal cannula The technic consists of connecting the outer end of the cannula with a microphone by means of a short piece of rubber tube The heart sounds are shorter and have vibrations of a lower frequency than when recorded from outside the chest

CONCLUSIONS

Phonocardiography has grown up slowly among the other graphic methods on account of technical difficulties Its importance is becoming greater from year to year In order to appreciate it one should keep in mind that ausculta

tion and phonocardiography are two inseparable technics for the same phe nomena Therefore whenever auscultation is useful, phonocardiography also is of help for the diagnosis However phonocardiography should not be considered merely as a way to check and confirm auscultatory findings On the one hand phonocardiography is more perfect than auscultation and is not hampered by the various technical failures of the human ear and the psychological limitations of the observer s mind On the other hand, having control of subsonic bands of vibrations phonocardiography is a more com plete method

The fields of cardiology which more commonly require phonocardiography are the following Rheumatic heart disease with valvular lesions luetic heart disease with aortic valve damage or aortitis calcific aortic stenosis, coarctation of the aorta congenital defects of the heart in general bacterial endocarditis adhesive and constrictive pericarditis, conditions associated with extensive myocardial damage, disturbances of the heart rate and rhythm

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CHAPTER 4

Tracings of the Slow Vibrations of the Chest

(CARDIOGRAPHY)

HISTORY

The first "cardiograms" were recorded at the apex Impressed by records of intracardiac pressure obtained by Chauveau and Marey Marey and Potain tried to record the motion of the heart by applying a capsule over the cardiac apex Good tracings are already reported in Marey s textbook in 1885 [8] It is interesting to note that in spite of the different technic and against any likelihood the first tracings presented some resemblance to those of Chauveau and Marey For many decades the tracing of the apex was considered typical only if it presented a positive plateau like wave during systole

The recording apparatus was at first a Marey s capsule Then a specially built box containing a spring and ending with a button was firmly pushed against one of the intercostal spaces (Fig 2) This cardiograph was connected with a Marey s capsule by means of a rubber tube

A second stage in the development of the instrument was represented by the use of a Marey s capsule connected with a Frank s capsule The mirror of the latter projected its light beam onto a photographic film which recorded the vibrations of the former (Cushney [1] Hess [2] Weitz,[10] Weber[9])

It was soon recognized that the cardiogram has different aspects in the sitting supine and left sided positions[6] The last position was considered the best by Pachon and Fabre[5] because it gives tracings which most resemble those of Chauveau and Marey

TECHNIQUE

Only the most recent and perfected methods shall be described

Linear Cardiography

The linear microphone[7] has been already described in detail (p 28) The chest piece is a funnel with a side tube (Fig 19) This is held in place by a stethoscopic microphone and is connected by a short rubber tubing with the linear microphone This set up has the advantage of recording simultaneously the low frequency vibrations (cardiogram) and the rapid vibrations (phono cardiogram) of the chest The cable of the linear microphone is plugged into *the outlet of an electrocardiograph The microphone with the chest piece is*



FIG 35 Technical set up for recording simultaneously a phonocardiogram and a cardiogram (high and low frequency tracings) *S* = stethoscopic microphone *L* = linear microphone

held in place by a rubber strap under moderate tension and the tracing is recorded during apnea (Fig 35)

The slow vibrations set up by the air in the funnel are transmitted to the linear microphone This transforms without distortion air waves of any frequency (Fig 13) into electrical pulsations which are recorded by the galvanometer of the electrocardiograph As the slow vibrations are thousands of times larger than the more rapid which have an acoustic value (p 30), there is no need for filtration or special magnification

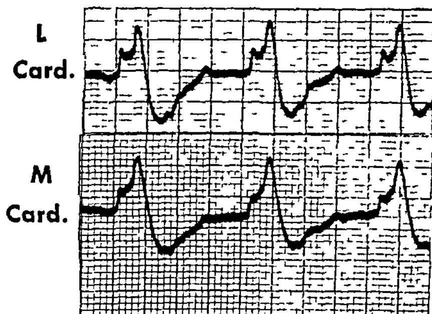


FIG 36 Low frequency tracing (apex cardiogram) *Top* recorded with a crystal microphone of a linear type (amplified) *Bottom*, recorded with the electromanometer (high sensitivity microphone)

Manometric Cardiography

The cardiogram can be recorded also by using an electromanometer as demonstrated by Johnston and Overy[8] The chest piece is that used with the previous method It is connected by a short piece of hard rubber tubing to the tube of the electromanometer The latter is provided with a high sensitivity microphone and the whole system is filled with air

Comparative studies by the author[5] have shown that only slight differences are present between tracings of linear and manometric cardiography (Fig 36) They consist of slightly larger rapid waves in the former, slightly larger slow waves in the latter

The electromanometer is more expensive requires several hours of warm

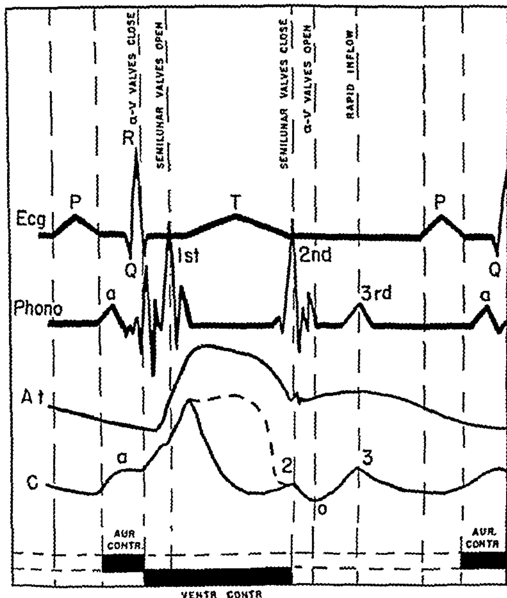


FIG 37 The two main variations of the low frequency tracing at the apex (cardiogram—*C*) compared with the electrocardiogram and phonocardiogram and an aortic tracing (*A t*)

ing is not easily portable and needs delicate adjustment before use For these practical reasons linear cardiography is still preferable to manometric cardiography

The cardiogram can be recorded over any area of the precordium as shown by the author[4] and in any decubitus That of the apex is called the *apex*

held in place by a rubber strap under moderate tension and the tracing is recorded during apnea (Fig 35)

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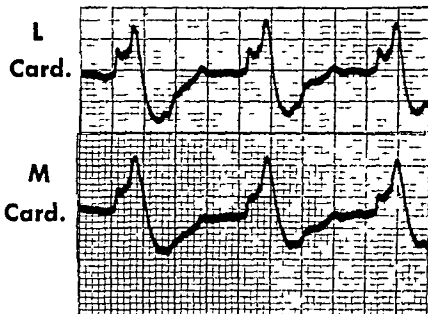


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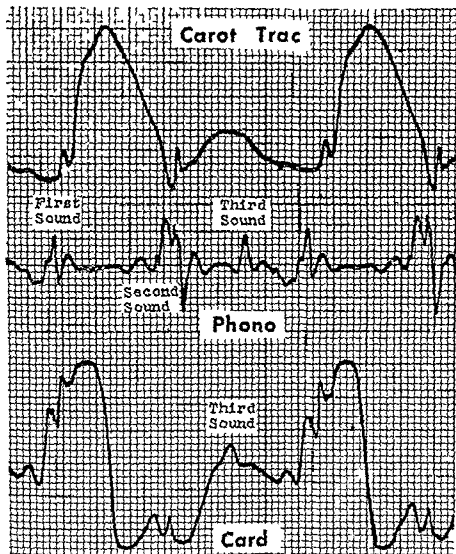


FIG 38 Low- and high frequency tracings (cardiogram and phono cardiogram) and carotid tracing of a normal subject (direct writing record) In this case the third sound has a slight delay over the wave of rapid filling of the apex cardiogram

closure of the a v valves (Figs 25 and 37) A subsequent notch frequently marks the opening of the semilunar valves (Notch 1b) Then the curve rises again at the beginning of ejection at the time of the rise of the carotid pulse

There may be two important variations from this scheme Sometimes the first peak is followed by a second which coincides with the opening of the semilunar valves In other cases a positive plateau is present during systole

On the other hand a deep inverted wave may occur during systole, changes of volume having predominance over effect of motion In such cases the curve rises after the systolic depression and reaches a peak at the time of closure of the semilunar valves (Point 2a) From this point on the various types of tracings become similar

Since diastole is accompanied by two phases of rapid inflow, there are two main diastolic waves in the cardiogram The first is represented by the wave of rapid filling during early diastole This is usually well defined and its peak is simultaneous with the third heart sound (Point 3) A deep depression (Point 2b) follows the Notch 2a and precedes the Point 3 The bottom of this depression occurs during the isometric relaxation period and coincides with (or precedes slightly) the v wave of the jugular and hepatic tracings (opening of the a v valves) After a few small undulations the second phase of rapid filling caused by atrial contraction, is marked by the small wave 4 as described above

REGIONAL CARDIOGRAMS

These tracings have been studied by the author[4, 11] in normal subjects (Fig 39) and in clinical cases Their interest was increased by the study of Johnston and Overy[3] with the electromanometer

PULMONARY AREA (SECOND TO THIRD LEFT INTERSPACES) The waves are usually small but amplification facilitates their study The cardiogram shows a relatively marked presystolic wave (*4*) and a well defined positive wave during early systole (Wave p) The former is probably due to the contraction of the left atrium transmitted through intermediate structures The latter is an arterial wave due to the pulsation of the pulmonary artery (Fig 39)

AORTIC AREA (SECOND TO THIRD RIGHT INTERSPACES) The various waves are small in normal subjects The cardiogram shows a well-defined wave during the tension period (Notch 1a) and a positive wave during early systole The former is transmitted from the left ventricle the latter is an arterial wave due to the pulsation of the ascending aorta (Wave p) Closure of the aortic valves is marked by a deep notch (2a) (Fig 65 *Asc Ao*)

TRICUSPID AREA (FOURTH TO FIFTH RIGHT INTERSPACES) The cardiogram shows a marked presystolic wave (Wave 4) small systolic notches (1a and 1b) and a well-defined early-diastolic wave (Wave 3) The first is apparently due to the contraction of the right atrium the latter is due to the rapid filling of the right ventricle

SUPRASTERNAL NOTCH A single large systolic wave (p) is recorded over this area This is caused by the pulse of the aortic arch transmitted through

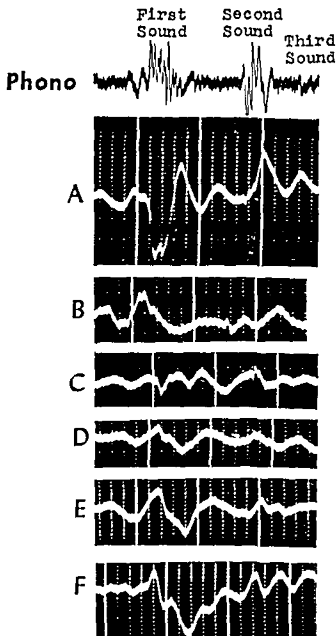


FIG 39 Low frequency tracings over various pre cordial areas (regional cardiograms) in a normal subject *A* apex *B* midprecordium *C* pulmonic area *D* Aortic area *E* tricuspid area *F* epigastrium

the structures of the upper mediastinum Closure of the semilunar valves (2a) is evident on the descending limb of the curve (Fig 65 *S Trac*)

In the writer s opinion appropriate symbols given to the various waves of the cardiograms would simplify their recognition (Fig 37) The rapid positive wave of rapid filling should be called *3* (third sound) The small wave present at the time of atrial contraction should be called *4* (fourth sound) The small wave terminating the tension period should be called *1a* the larger one terminating the ascending phase *1b* The small rebound, ending systole should be called *2a* The important drop which precedes rapid filling should be called *2b* it marks the opening of the a v valves Lastly, the wave of arterial type which may be recorded over the aortic and pulmonary areas should be called *p* (pulse)

CONCLUSIONS

Cardiography has lost much of its importance for the diagnosis of arrhythmias of the heart having been replaced by electrocardiography It still has a place in the study of valvular and septal defects Interesting cardiographic data may be found in cases of hypertensive or coronary heart disease or cor pulmonale In all of them cardiography may be used in order to recognize the phase and significance of the sounds recorded by phonocardiography The value of cardiograms for recognition of diastolic sounds is great and superior to that of jugular tracings Cases of adhesive pericarditis and of aortic aneurysm often require an accurate cardiographic study

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CHAPTER 5

Tracings of the Pulsations of the Epigastrium

(EPIGASTRIC TRACINGS EPIGASTRIC CARDIOGRAPHY)

HISTORY

Epigastric tracings were recorded early by students of cardiology MacKenzie[6] described this tracing, Lang[3] and Dressler[1] analyzed its data with care The most detailed study however was made by Fukui[2] in 1928 Interest in the study stemmed mainly from the possibility of recording movements of *the right heart through the diaphragm However pulsations of the* left ventricle the liver, and the abdominal aorta have to be taken into consideration

The usual three stages in the instrumentation took place first a Marey's capsule tracing graphs on smoked paper then a Frank s capsule with mechanical photographic recording, later the author used a crystal microphone with linear response and obtained electro optical records[4, 5]

TECHNIC

Present day technic is similar to that employed for cardiography (p 64) and is based on the use of the linear microphone A rubber strap tied around the upper part of the abdomen and placed under tension holds a microphone with a chest piece sunk into the epigastric triangle (Fig 54) The tracing is

recorded during apnea An epigastric phonocardiogram an apex phono cardiogram or an apex cardiogram should be recorded with the epigastric tracing A second rubber strap holds a second microphone over the apex (linear in the case of the apex cardiogram stethoscopic in that of the phono cardiogram)

CAUSE OF THE WAVES

The following factors should be considered in analyzing an epigastric tracing

1 Pulsations transmitted through the diaphragm from the heart mainly the right heart,
2 Effect of changes of intrathoracic pressure on the diaphragm
3 Pulsations of the liver
4 Pulsations of the abdominal aorta

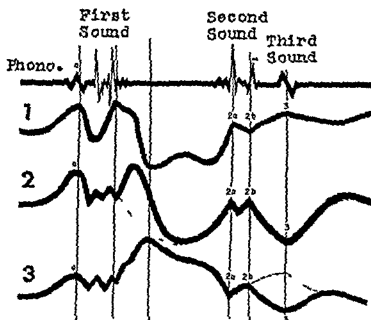


Fig 40 Scheme of the various types of epigastric tracing

VARIOUS TYPES OF EPIGASTRIC TRACINGS

Epigastric tracings are by no means uniform An amazing variety of patterns can be obtained even in different normal individuals However the tracings can be classified into four groups two of them common the others less so As shown by Fig 40 certain parts of the tracing are similar in all while others vary

A high positive wave is always present during presystole It should be

called *4* (fourth sound) During the period of tension the curve either falls suddenly below the base line or has two small notches caused by the two valvular events of early systole They should be called *1a* and *1b*, as on the apex cardiogram A positive wave may be present in the phase immediately following However most tracings exhibit either a wide negative plateau during most of ventricular systole (Fig 41), or a positive wave simultaneous with the arterial pulse (Fig 55) Closure of the semilunar valves is marked on all records by a well defined notch, which may be either upright or inverted and should be called *2a* The opening of the a v valves is well defined in most records and should be called *2b* The rapid filling of the ventricles is also indicated by a large wave which may be either upright or inverted and should be called *3* (third sound)

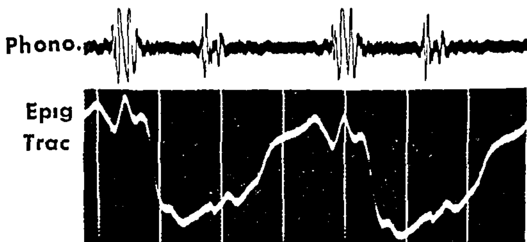


FIG 41 Phonocardiogram over midprecordium and epigastric tracing

The presystolic wave *4* is largely due to the mechanical movement of the right atrium transmitted through the diaphragm The two notches *1a* and *1b*, when present, are caused by the valvular events of early systole and probably *more by those of the right heart The high systolic wave represents* the beginning of a systolic plateau abruptly interrupted by the following systolic collapse It is probable that the plateau is caused by motion of the right ventricle, the collapse by volume changes of this chamber and by changes of intrathoracic pressure The high wave of other tracings on the contrary occurs later and is of arterial type It is probably a transmitted aortic pulsation

Four different types of tracings can be recognized[4] (Fig 40) Type 1 tracings show influence of ventricular events during systole as well as during

early diastole Type 2 tracings show a definite predominance of waves due to changes of intrathoracic pressure over waves due to movements of the heart Type 3 tracings are largely influenced by the aortic pulsation but still show effects of motion during presystole and early systole effects of thoracic pressure changes during diastole Type 4 tracings are pure liver tracings and will be described later (p 104) They occur when the enlarged liver occupies the epigastrium

CONCLUSIONS

If it is true that the right heart has a particular influence on the epigastric tracing *information about dynamics of the right atrium and ventricle will be* obtained by the study of this record Simultaneous (or immediately subsequent) tracings of the apex and the epigastrium complete therefore our knowledge of the dynamics of the two halves of the heart The atrial wave of the tracing is larger in the epigastric tracing than in any other record of cardiac pulsations In cases where a hepatic tracing is necessary this should always be compared with the epigastric tracing in order to avoid confusion between actual liver pulsations and transmitted pulsations

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CHAPTER 6

The Atrial Wave in the Various Tracings

The importance of the problem is self evident If the atrial wave occurs at different times in tracings recorded over different areas of the chest this means that some of the tracings are affected more by right atrial contraction and others more by the left

The interval between right and left atrial contractions is so small in normal individuals that very little difference in the time of appearance of the waves is to be expected Even so a marked difference can be found between tracings of the apex and epigastrium On the contrary patients with fibrosis of the atrial myocardium are more apt to give evidence of increased delay between right and left atrial contraction Studies made on such patients[1] have established the following data

1 Tracings recorded over the second right interspace frequently show left atrial contractions as a downward wave Tracings recorded over the second left interspace frequently show it as a high positive wave (Fig 39C)

2 Tracings of the apex usually show a positive wave for left atrial contraction sometimes preceded by a negative wave for right atrial contraction (Figs 39A 66 221, 222)

3 Tracings of the epigastrium show mainly the effect of right atrial contraction which is revealed by a high positive wave (Fig 42) Sometimes a negative phase follows this at the time of left atrial contraction (Figs 41 221 222)

The atrial wave (Wave 4) presents good development and an unusual

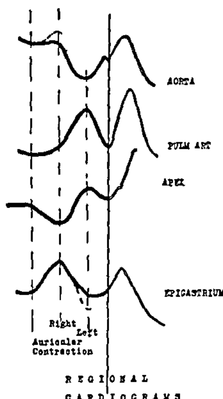


FIG 42 The atrial wave (black) in the regional cardiograms and in the epigastric tracing

height in certain types of patients A large atrial wave in the second and third left interspaces is found in most cases of rheumatic heart disease with mitral lesions or mitral plus aortic defects A large atrial wave in the epigastric tracing is found in cases of cor pulmonale

In cases of a v block the atrial waves are high both at the epigastrium and at the apex In all of them the atrial waves are far more pronounced in the low frequency tracings than in the phonocardiogram

CONCLUSIONS

The tracings of the apex and epigastrium show the effect of presystolic filling of the respective ventricle while the waves recorded at the base are due to indirect transmission of the atrial waves and the filling of the opposite ventricle sometimes becomes apparent as a negative wave It is likely that the waves recorded in the areas of the base are due to direct motions of the atria In particular motion of the left atrium can be transmitted forward with a move

ment of rotation which pushes the lung or the pulmonary artery against the chest wall, and acts in the opposite way on the aorta

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SECTION B

Vibrations of the Body Due to Cardiovascular Action

CHAPTER 7

The Ballistocardiogram

HISTORY

The first study of ballistocardiography was made by Gordon[4] in 1877 He suspended a bed from the ceiling by ropes and obtained a record of its motion determined by the heart beat Henderson[9] in 1905 built an elaborate sus pended table and obtained records which he tried to correlate with cardiac output In 1913 Satterthwaite[19] obtained tracings from a patient sitting on spring scales In 1922 Heald and Tucker[8] obtained records from a suspended platform by using an electrical method In 1933 Abramson[1] obtained good tracings from a specially built chair

More recent studies were accomplished by Nickerson and Curtis [15] Starr [20] and Dock [3] while most of these studies were concerned with application of the method to determination of cardiac output others tried to ascertain the time relationship of the various waves Among them the most important are those of Dock and Taubman [3] Hamilton and co workers [7] Gubner and co workers [6] Nickerson [14] De Lalla [1] and Jones and Goulder [10] A subsequent study was published by the author with Contro [13]

TECHNIC

A low frequency critically damped apparatus has been advocated by Nickerson [16] This instrument has the advantage that all waves are the result

of forced movements, so that phasic reinforcements and damping does not occur

In the photoelectric type of apparatus advocated by Dock [3] the body is free to move It might be said that this reproduces the conditions of the free moving table originally built by Henderson [8] However, the set up is damped by the natural period of the tissues and by attrition with the table Therefore

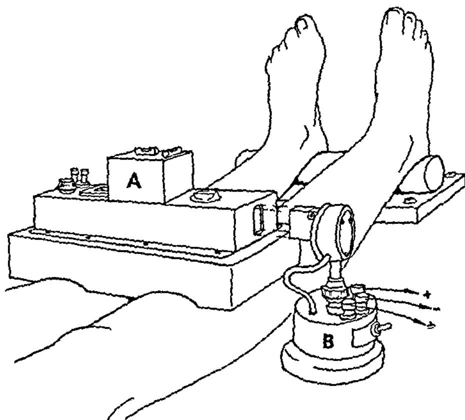


FIG 43 The photoelectric ballistocardiograph *A*, crosspiece *B* photocell unit

while this type of instrument is somewhat similar to the original undamped table of Henderson, no artificial swing is added to that of the body

Comparative tests between the electromagnetic apparatus also used by Dock,[3] the piezoelectric apparatus of Sheehan [10] and the photoelectric apparatus of Dock [3] have been made by Gubner [5] He concluded that when respiration is suspended the photoelectric device without filter gives tracings closely resembling those of the electromagnetic set up However, he found a minimal time lag in the former due to the fact that it records displacement instead of acceleration *

* *It should be kept in mind that the swinging table measures acceleration the electromagnetic system measures velocity and the optical system measures displacement*

Photoelectric Apparatus

The photoelectric apparatus (Fig 43) consists of (1) A *cross piece* of light wood containing the source of light and placed transversely over the legs of the patient (2) A phototube contained in a separate unit and so placed that the light beam enters the opening this is revealed by a prism projecting the light on a frosted glass at the top of the unit

The phototube is connected with the galvanometer of an electrocardiograph by means of two wires

Accurate records are obtained only with a completely immobile table and with a light beam absolutely perpendicular to the phototube Therefore cer tain precautions are currently followed in the author s laboratory

1 A wooden board 1 inch thick is placed over the examination table and is firmly pushed against the wall

2 A small rectangular board with four regulating screws supports a wooden cylinder which is placed under the ankles of the patient

3 The cross piece rests over the patient s ankles This piece contains the source of light covered by a slitted surface Two bubble levels at right angles on the cover of the latter determine whether the cross piece has any inclination in a transverse or longitudinal direction Adjustment of the screws permits compensation for any angle resulting from the curvature of the tibiae of the subject

4 The phototube is placed very near to but not touching the cross piece It is moved until a shadow caused by the edge of the slit forms exactly in the center of a window of this part of the apparatus It is raised until no trans verse shadow appears at the bottom

5 A low firm pillow supports the head of the subject without touching neck or shoulders The subject s hands are placed on the lower quadrants of the abdomen

6 If the waves are small and in case of special studies magnification is obtained by means of a specially built preamplifier The degree of magnifica tion is so regulated that the waves do not reach the limits of the film strip

When the details of the tracing are considered satisfactory the subject is invited to hold his breath in an intermediate position and the tracing is re corded in apnea It is also possible to use an electrical filter and to record the tracing with normal respiration In such a case some distortion of the waves is unavoidable

The tracings can be recorded by a direct writing electrocardiograph (San born Poly Viso) at speeds of 10 25 or 50 mm per second or on photo graphic film (Sanborn Stetho Cardiette or Twin Beam) at film speeds of 10 25 or 75 mm per second

Calibration may be obtained by hitting the foot of the bed (in Starr s

method) or the head of the patient (in the case of the magnetic or photo electric methods) with a known force It is preferable to place a small scale, exerting a known force (600 Gm) against the head of the patient then release it suddenly There should be an upward displacement of 20 mm in the tracing *If a greater or a lesser displacement takes place proportional com* pensation should be introduced in the calculations

Determination of the stroke volume by means of the ballistocardiogram has been attempted According to Paine and Shock [17] this can be done in the following way A baseline is drawn from one diastolic phase to the next The area of both the *I* and the *J* waves is approximated by assuming each wave to be a triangle and measuring the altitude of each from apex to base The average areas of these waves are then introduced into formula (1)

$$\textit{Stroke volume}\ (\text{cc /min}) = 100\sqrt{(2\ I\ \text{and}\ J)\sqrt{C}} \qquad (1)$$

where *C* represents the duration of a cardiac cycle in seconds

A cardiac index then is found by formulas (2) and (3)

$$\textit{Cardiac output}\ (\text{L /min}) = \frac{\textit{Stroke volume} \times \textit{Pulse rate}}{1000} \qquad (2)$$

$$\textit{Cardiac index}\ (\text{L /min /M}^2) = \frac{\textit{Cardiac output}}{\textit{Body surface area}\ (M\ )} \qquad (3)$$

The mean basal cardiac index was found[18] to be 3 35 L /min /M ^{2}, a value which is very similar to that of 3 30 ascertained by right heart catheterization

According to Starr the amplitude of the ballistocardiographic waves is *more closely related to cardiac strength* than to cardiac output

Determination of cardiac strength is made as follows [6] One of the smallest and one of the largest complexes is selected as typical and the vertical depths of the *I* and *J* waves from the baseline are measured in millimeters * The sum of these measurements e g $I+J+I+J$ is used and compared with that expected on the base of the equation

$$I+J+I+J\ \text{mm} = 23\ 6\ (\textit{subject s surface area in sq M}\ ) - 13\ 35$$

The difference between the expected value and that found in the tracing represents cardiac strength in percentage of norm

If the surface of the patient is 1 80 *sq* M , 23 6(1 80) − 13 35 = 29 13 is the expected figure If that actually found was 2+5+4+7 = 18 then 18 − 29 13 = −11 13 and the deviation from the expected value is

$$\frac{-11\ 13}{29\ 13} = -38 \text{ per cent}$$

* If calibration with 600 Gm displaces the tracing more or less than 20 mm the measure ments should be corrected in proportion

DESCRIPTION AND INTERPRETATION OF THE WAVES

The ballistocardiogram (bcg) consists of the rapid succession of positive and negative waves the time relationship of each peak is easily ascertained*, on the other hand only the first branch of a wave should be considered, the second branch being already the main component of the following wave (Fig 49) A certain difference in the time relationship of certain waves may be found between the various subjects This is partly due to the length of diastole more waves are present when diastole is long less waves when it is short

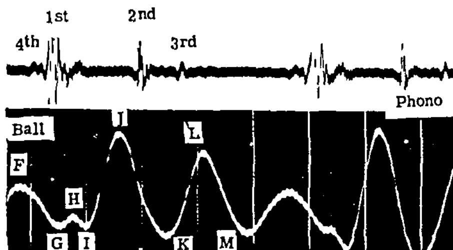


FIG 44 Ballistocardiogram and phonocardiogram of a normal subject

The most useful graphic tracings for the interpretation of the bcg are the phonocardiogram and the arterial tracings while the electrocardiogram the phlebogram and the apical cardiogram are of help only in special cases

F WAVE The beginning of the normal cardiac cycle can be placed near the beginning of the first sound complex This point should be called F[13] In several subjects this point occurs from 0 02 to 0 04 second before the first vibration of the first sound (Fig 44)

G WAVE The first part of this small negative (caudad) wave is made of a descending branch which starts at or slightly before the beginning of the tension period of ventricular systole and is simultaneous with the initial part of the first sound complex[12] The peak of G takes place at the time of that large vibration of the first sound which marks the closing of the a v valves

* This description of time relationship can be used in the interpretation of tracings recorded with the table and of those recorded with the magnetic or photoelectric systems It cannot be used in the interpretation of tracings recorded with the Nickerson system because its damping device causes a remarkable distortion of the waves and a marked time lag

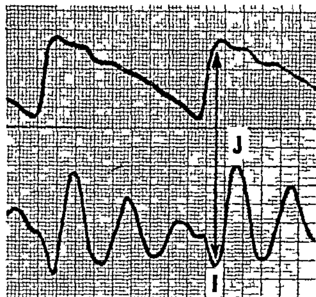


FIG 45 Ballistocardiogram and carotid tracing

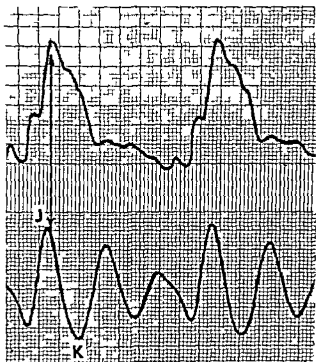


FIG 46 Ballistocardiogram and tracing of the abdominal aorta

(p 44) It occurs before the rise of the pulse in the tracings of the suprasternal notch and of the carotid or subclavian arteries

H WAVE The ascending branch of this small positive (cephalad) wave takes place during the central part of the first sound complex Its peak coincides with

1 That large vibration of the first sound which marks the opening of the semilunar valves (p 44)
2 The beginning of flow within the ascending aorta revealed by a notch in the suprasternal tracing (p 128), the rise of the carotid pulse and the rise of the c wave of the jugular tracing (p 99)

I WAVE The descending branch of this large negative (caudad) wave starts at the beginning of ejection together with that vibration of the first sound which marks the opening of the semilunar valves (Fig 44) Its negative peak coincides with

1 The peak of the c wave of the jugular tracing
2 The peak of the pulse of the suprasternal notch and the carotid arteries (Fig 45)
3 A small negative wave of the abdominal aortogram which precedes the main wave (Fig 46)

J WAVE The ascending branch of this large positive (cephalad) wave takes place during the first half of ventricular ejection The peak precedes considerably the second sound It takes place at the time of

1 The peak of the abdominal aortogram (Fig 46)
2 The systolic depression of the jugular tracing
3 The peak of the femoral pulse

K WAVE The descending branch of this deep negative (caudad) wave takes place during the end of systole and during the isometric relaxation period Therefore it rides over the main vibration of the second sound at least in normal subjects Its negative peak *is sometimes sharp* sometimes broad It is simultaneous with a small vibration of the second sound complex which marks the opening of the a v valves (p 50) It should be simultaneous with the v wave of the jugular tracing however this wave is sometimes slightly delayed and may not coincide with it It follows the peak of the main wave in the abdominal aortogram It coincides with the peak of the tibial pulse and slightly precedes that of the pedal pulse (Fig 47)

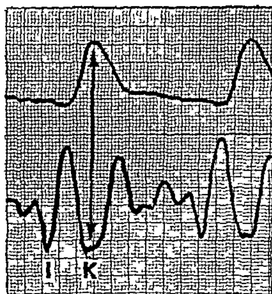


Fig 47 *Ballistocardiogram and pedal tracing*

L WAVE The ascending branch of this positive (cephalad) wave takes place in early diastole during the rapid passive filling of the ventricles Its positive peak follows the third sound or a ventricular type of triple rhythm (p 422) by a short interval (0 04–0 06 second) (Fig 44) It takes place during the dicrotic wave of the pulse in the aortic arch as revealed by suprasternal and carotid tracings It coincides with the predicrotic notch of the abdominal aortogram There is no coincidence with the waves of the jugular tracing or with the pulse of the lower extremities

M WAVE This negative (caudad) wave is variable in shape and is markedly affected by the heart rate Its descending branch takes place during mid diastole Its negative peak may coincide with the dicrotic wave of the abdominal aorta and of the tibial arteries (Fig 46) On the other hand there may be a single wide negative wave with a peak at the time of the fourth sound ($M+O$)

N WAVE This wave when present, is small It may be only a rebound of the previous M wave

O WAVE *When there is bradycardia and N is present, it may be followed by* a small negative wave In other cases, M and O are fused in one

The beginning of the cycle can be placed usually at the point F The need for using this new letter is due to the fact that more details are revealed by the tracing when using high speed film and an amplifier [13]

The main complex of the ballistocardiogram is the triphasic wave H I J K L The interpretation of this has been firmly established by Hamilton and Dow[7] and by Starr[20] (Figs 48 and 49) It is based on the following principles

1 *Any acceleration of the blood in the main arterial channels is accompanied* by a movement of the body in the opposite direction any deceleration of the blood is accompanied by a movement of the body in the same direction

2 Rapid ejection causes displacement of a large mass of blood first in a headward direction (ascending aorta), then in a footward direction (descending aorta)

3 The braking effect of the branchings and smaller vessels particularly in the lower extremities results in a deceleration

In patients with a v block the atrial wave of the jugular tracing coincides with a negative (footward) wave of the bcg This wave has been called X[13] It can be explained by the arrest of venous flow by the atrial contraction This causes a deceleration of the entire venous circulation and a footward movement of the body

In most normal subjects either of the following possibilities can take place

1 There is a large negative diastolic wave (M–O) which lasts through presystole The fourth (atrial) sound occurs during this wave

2 There is a small positive wave (F) which coincides with the fourth (atrial) sound

It is likely that in most cases the atrial contraction contributes to the G wave Both the atrial contraction and the initial raising of the a v valves caused by ventricular systole arrest the venous flow (deceleration) and favor a footward movement of the body As this deceleration follows the atrial contraction the G wave takes place during early ventricular systole

The H wave takes place during the second half of the tension period of ventricular systole At this time the contraction of the papillary muscles lowers the raised leaflets of the a v valves and accelerates the venous flow into the atria The footward movement of the blood is apparently accompanied by a small headward movement of the body

The I wave coincides with the rapid ejection of blood into the ascending aorta and pulmonary artery As this is in a headward direction the body moves suddenly in a footward direction

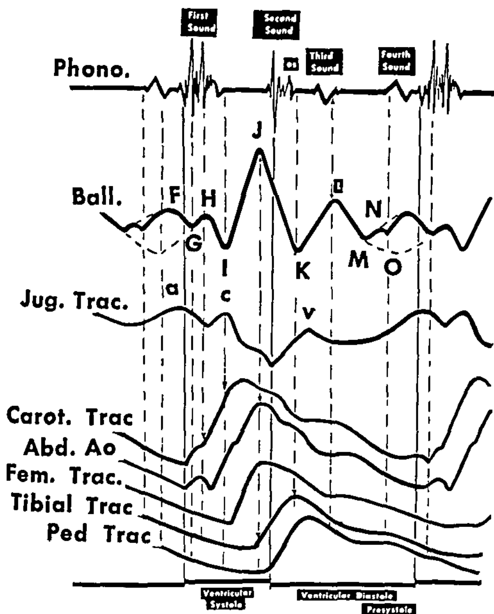


Fig 48 Scheme of the time relationship of the waves of the ballistocardiogram with those of other tracings

The J wave takes place during the second phase of ventricular ejection The pulse wave has rounded the aortic arch and is moving rapidly toward the lower extremities Therefore, the body makes a headward movement The end of this wave takes place before the second sound at a moment where

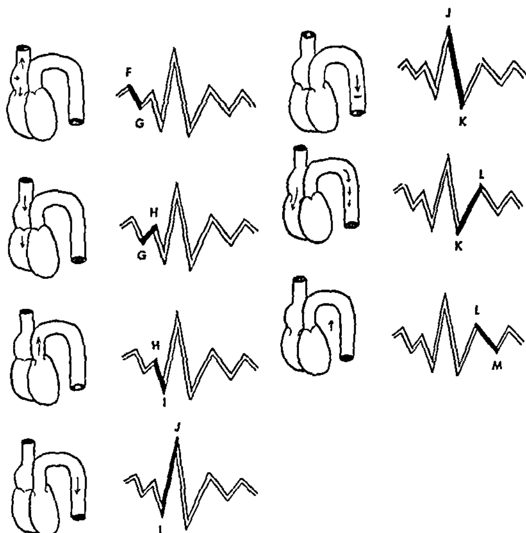


FIG 49 Cause of the various waves of the ballistocardiogram

braking of the blood starts in the vessels of the lower extremities The coincidence of the point J with the peak of the abdominal and femoral pulse is extremely significant in this regard The braking or deceleration of the blood causes a footward movement of the body There are several reasons for a sharp end of this movement One is the opening of the a v valves revealed by a small *vibration in the sound tracing and slightly preceding the peak* K

the onrush of blood into the ventricles contributes to end the previous movement of the body Another is that sudden reversal of velocity of the blood which precedes the dicrotic wave in the abdominal aorta and femoral arteries

During the tract K–L the blood of the ascending aorta has rebounded over the closed semilunar valves and moves forward into the descending aorta At the same time the rapid passive filling of the ventricles has reached its maximum as proven by the occurrence of the third heart sound Both the rapid filling and the dicrotic wave in the abdominal aorta contribute to a headward movement of the body

The end of the dicrotic wave in the abdominal aorta and in the arteries of the lower extremities coincides with the point M Whenever there is bradycardia this point is sharp and visible Otherwise there may be fusion with the G wave of the following cycle

The N wave when present is probably only a rebound or after oscillation of the body The slow accumulation of blood in the atria resulting in a deceleration of the venous flow may contribute to it

TIME INTERVALS

These intervals have been particularly studied by Jones and Goulder [10] It should be kept in mind that they have been determined with a low frequency critically damped apparatus Important differences exist between these data and those found with other apparatus

Q–I interval 0 15–0 195 second
Q–J interval 0 29–0 37 second
Q–K interval 0 49–0 57 second

RESPIRATORY VARIATIONS

Respiration modifies considerably the height of the various bcg waves Respiration affects the tracing by changing the position of the heart in relation to the long axis of the body and by changing venous return to either ventricle It is typical to observe an increased height of the I and J waves in inspiration and a decrease in expiration [11]

CONCLUSIONS

If the accuracy of calculations for measuring cardiac output or cardiac strength is confirmed ballistocardiography represents an easy and simple way for determination of these values Even if absolute values should be found slightly inaccurate variations of the values in any one case would still be of great clinical interest

The patterns revealed by the tracing are of value in coarctation of the aorta and atherosclerosis of the aorta Abnormal patterns are present in coronary heart disease, peripheral arteriosclerosis bundle branch block and heart failure Future experience shall prove whether or not these patterns are more typical or more easily detected than with other graphic methods

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SECTION C

Venous Pulsations

CHAPTER 8

Tracings of the Pulsations of the Jugular Veins

(PHLEBOGRAPHY JUGULAR TRACINGS)

HISTORY

The first tracings of the jugular veins were recorded in the dog by Wedemeyer in *1828* [27] Friedreich[6] *recorded venous tracings from the neck of* patients in 1865 Two years later, Potain recorded simultaneous tracings of the apex beat and of the carotid radial and jugular pulsations His interpretation of the tracings was accurate even in the light of present day knowledge His studies were followed by those of Mosso [18] Riegel [24] François Frank [4] Gottwalt [9] Gerhardt [7] and Fredericq [5] MacKenzie[16] initiated a clinical and physiologic evaluation of the jugular tracing soon followed by Wenckebach[29] and many others

The method has been subsequently considered of secondary importance on account of the widespread adoption of electrocardiography However *interest* was shown by numerous South American studies which have appeared in the last twenty years from Houssay[12] to Caeiro [1]

Several stages can be recognized in the development of the method The first method was based on the light application of a funnel over the bulb of the right jugular vein This was connected by air transmission with a Marey

capsule which transcribed the waves on smoked paper Necessity of avoiding any even slight compression of the vein led to the development of several ingenious devices such as a little ball of sambucus moving a delicate lever [19, 20]

A second method was based on the use of an optical photographic recording This was obtained first by mechanical optical transmission (lever to mirror funnel to Frank s or Wiggers Dean s capsule) Later a small mirror reflecting a beam of light was pasted over the skin above the jugular vein A subsequent ingenious device was based on the principle of interposing the patient directly between the source of light and the camera [21]

The most recent development consists of a special applicator and a crystal microphone having a linear type of response [17]

TECHNIC

Good records may be obtained from the jugular vein of any normal person if the venous pressure is above the atmospheric Therefore in normal subjects the vein should lie slightly below the level of the right atrium while in patients the veins may be above this level The patient should be placed in the supine position if necessary his pelvis can be raised or his head lowered (subjects with low venous pressure) In the first case venous return and venous pressure are increased in the second venous pressure is increased by gravity because the jugular vein is lower than the right atrium In cardiac patients with increased venous pressure good tracings can be recorded in a semi recumbent position occasionally even in the sitting position

A different technic has been advocated by Groedel [10] A piece of metal weighing 100–300 Gm is placed over the applicator in order to exclude the slow waves mainly caused by changes in flow (tidal waves) The resulting tracing is supposed to reveal only the concussion waves The latter are multiple pressure waves of small amplitude occurring both during systole and diastole on account of the various events of cardiac dynamics [10] This technic is not commonly accepted and might be used only for special studies

The patient should be instructed to hold his breath in an intermediate position during the recording

The applicator is a double rim circular chamber or cup having an internal diameter of 20 mm and a depth of 5 mm * (Fig 50) The outer chamber is connected with a rubber bulb and is used to insure adhesion to the skin without pressure After squeezing the bulb the applicator is gently applied over the skin then the bulb is released in order to create the necessary suction The inner chamber communicates with a linear microphone through a small hole and a short rubber tube The venous pulsations set up changes of pres

* This is supplied by the Sanborn Co of Cambridge Mass or by the Cambridge Instrument Co of New York

sure in the cup, the linear microphone transforms them into equivalent electrical pulsations The electrical waves are then recorded by an electrocardiograph after amplification Both the photographic and the direct writing types of electrocardiographs can be used Speeds of 50–75 mm /sec are necessary for a good analysis of the tracings

In patients with small waves a preamplifier can be placed between the microphone and the galvanometer

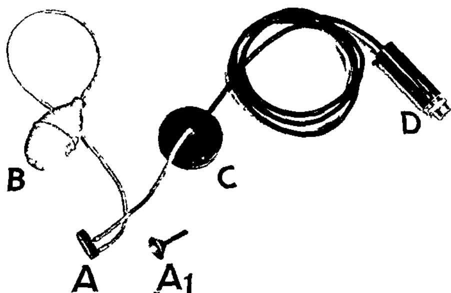


Fig 50 Linear microphone for recording jugular and carotid tracing *A* suction applicator for jugular tracing A_1, funnel for carotid tracing, *B* rubber bulb *C* linear microphone *D* plug connecting with the electrocardiograph (From Rappaport and Sprague Courtesy of the C V Mosby Co)

It is self evident that the venous tracing varies according to the natural frequency of the recording instrument Present day instruments have an over all natural frequency higher than 100 per second, therefore modern tracings are accurate and reliable even though sometimes slightly different from those recorded in the past

A different device has been used by the author in certain cases The cuff of a special blood pressure recorder (p 154) is wrapped around the neck A slight pressure (5–10 mm Hg) is used in order to insure a good contact Then the tracing is recorded by means of a linear microphone Even though the tracing frequently presents a strong arterial component, the atrial waves of the jugular tracing are well marked

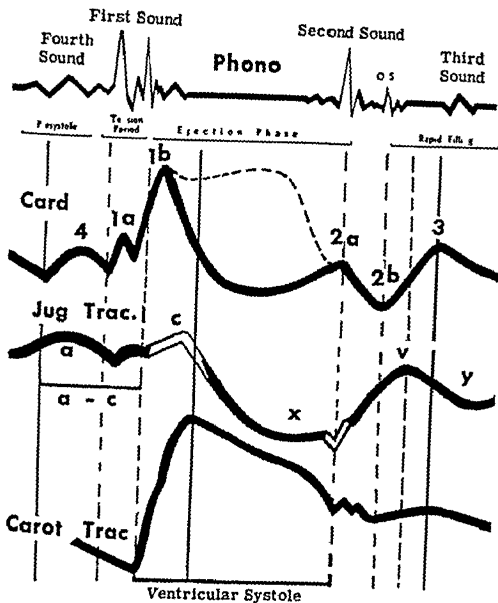


FIG 51 The jugular tracing compared to other mechanical and sound tracings In black that part of the jugular tracing which has a venous origin In white that part which is transmitted from the arterial system

Good tracings may be obtained from the entire jugular vein, if it is engorged. Otherwise the best tracings are obtained from the *jugular bulb* which lies slightly above and about one inch external to the sternal end of the clavicle.* In general the right jugular vein is preferred, being nearer to the right atrium. However in certain cases good tracings are obtained from the

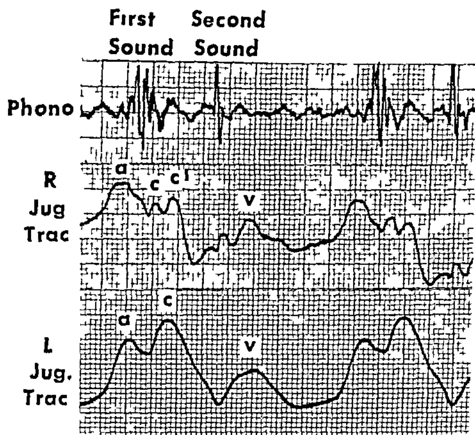


Fig 52 Jugular tracings of the right (*R Jug Trac*) and the left (*L Jug Trac*) side of the neck. Note the precession of the *a* wave and the double *c* wave on the right side. The wave *c* is of tricuspid origin while *c*[1] is due to an arterial impact.

suprasternal notch or from the left jugular vein. It should be noted that remarkable differences between the jugular tracings of the two sides may occur[6] in regard to timing and shape of the waves (Fig 52).

ANALYSIS OF THE WAVES

If the patient is allowed to breathe normally the jugular tracing exhibits a slow and deep swing of the base line which is due to respiratory variations of

* In tricuspid stenosis Puddu[23] obtained high "a" waves over the jugular bulb, much smaller waves on a more distal section of the vein.

venous pressure Inspiration is accompanied by a drop of the base line and by larger pulsations expiration by a rise and smaller pulsations (Fig 78)

Following MacKenzie's description the jugular tracing presents three positive waves (a c and v) and three negative waves (the last two were called x and y) (Figs 51 52 53 and 57)

PRESYSTOLIC WAVE The presystolic wave (a) is positive and occurs during and after atrial contraction It is usually a rounded wave followed by a sharp drop during the tension period of the ventricles However, tracings recorded through an amplifier frequently present a tall and peaked presystolic wave

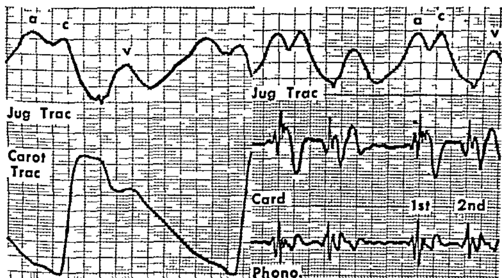


FIG 53 (*Left*) Comparison of the jugular tracing with the carotid tracing (*Right*) Comparison of the jugular tracing with a low frequency tracing of the apex (*Card*) and a stethoscopic phonocardiogram (*Phono*)

SYSTOLIC WAVE The systolic wave (c) is positive and occurs during early systole after the opening of the semilunar valves It is a peaked wave followed by a long and slow depression (*x* or systolic collapse) which lasts through most of the ejection period and slightly beyond it Toward its end a small notch may mark the closure of the semilunar valves

EARLY DIASTOLIC WAVE The early diastolic wave (v) is also positive It is a peaked wave which coincides with the opening of the a v valves and marks therefore the beginning of ventricular filling This is followed by a depression (*y* or diastolic collapse) When diastole is long another small positive wave (h) may be found after v (Gibson[8] Hirschfelder[11])

Two types of jugular tracings are most commonly recorded in normal subjects[30]

1 The *atrial pressure type* Here the c wave is characterized by a sharp decline at the onset of systolic ejection and during the traction on the atrio ventricular septum, just as in atrial pressure curves

2 The *modified arterial* type The c wave is high and rapid, due to impact of an adjacent artery It often resembles the first part of a central arterial pulse Tracings recorded with the cuff are usually of this type

MEANING OF THE WAVES

It has been shown[1] that each wave of the venous tracing is the result of changes of volume and of changes of pressure and velocity The relation be tween these three factors (volume, pressure and velocity) varies from moment to moment It seems likely moreover that in high venous pressure, changes of pressure predominate over changes of volume because the veins are already distended

The presystolic (a) wave is undoubtedly related to the contraction of the *right atrium as shown by the fact that it is absent in cases of atrial fibrillation* Its beginning marks the beginning of atrial contraction its peak the end of the same phase The a wave is largely a pressure wave[1] with additional volume and velocity components Actual regurgitation of blood is minimal in normal subjects, but may become more important when venous pressure is high and the superior cava is dilated The wave occurs about 0 10 second after the corresponding rise of pressure within the right atrium [13] The wave is much larger when simultaneous contraction of the atria and ventricles leads to im portant regurgitation of blood into the venous system [8, 9] as in nodal rhythm nodal premature beats and a v block

The systolic (c) wave is a pressure wave [1] In many cases it is due to transmission of the strong pulsation of the underlying subclavian or carotid artery and the wave is an unavoidable artifact In other cases the wave is venous and is caused by an arterial pulsation transmitted from the ascending aorta to the superior vena cava The same is true of the small notch marking the closure of the semilunar valves (Fig 52) It has been shown[3, 15-5] that occasionally a double (c) wave may be seen the first component being due to closure of the tricuspid valve the second to arterial pulsation (Fig 52 right phlebogram)

The early diastolic wave (v) is related to the opening of the tricuspid valve The rise preceding its peak is the expression of the gradual filling of the right atrium Therefore the (v) wave is chiefly a volume wave [1] However as the fall of the curve is caused by the opening of the tricuspid valve the descending limb is almost exclusively due to a drop in pressure [1]

A depression occurs during the tension period of the ventricles Its depth is greater when the interval between atrial and ventricular contractions is

longer The depression (x systolic collapse) occurs during the ejection phase of ventricular systole It is due to the suction caused on the venous system by the outflow of blood from the thorax and by the downward movement of the a v septum It is, therefore a wave due to increased speed of the blood and decreased volume of the vein

The depression (y) (diastolic collapse) follows the v wave Slow filling of the right atrium terminates this depression Sometimes it is followed by a small positive wave (h) which has been attributed to floating of the tricuspid valve

There is no constant relation between the waves of the phlebogram and those of the electrocardiogram, except for the time relationship between the intervals a c and P Q

The waves of the jugular and hepatic tracings practically coincide except for the c wave which is nearly absent in the latter

The fourth (atrial) sound slightly precedes the peak of the (a) wave of the venous tracing This coincidence is due to the fact that both events follow the atrial contraction, the fourth sound is due to the blood hitting the ventricular wall, it takes some time for the transmission of the (a) wave from the right atrium to the vein The descending limb of the (a) wave is so slow that it ends after the start of the first sound

The main vibration of the second sound complex precedes the peak of (v) by about 0 106 second while the third sound falls during the descending limb of (v), e g 0 064–0 078 second later [1] However there may be a much shorter interval between (v) and the third sound and there may even be coincidence This fact due to occasional slower transmission of the v wave reduces the importance of the jugular tracing in the identification of an early diastolic sound [14]

CONCLUSIONS

The jugular tracing is of definite value (1) in valvular defects chiefly tricuspid lesions (2) in congestive failure with functional insufficiency of the tricuspid valve (3) in bundle branch block (4) in septal defects (5) in cases with low voltage of the P wave of the electrocardiogram or where the existence of this wave is doubtful (6) in atrial flutter

The jugular tracing gives information concerning mechanical events of the right heart In this respect it still holds a special place together with the hepatic and epigastric tracings among graphic methods



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C H A P T E R 9

Tracings of the Pulsations of the Liver

(HEPATOGRAPHY HEPATIC TRACINGS)

The hepatic tracing is recorded easily whenever the liver is enlarged or lowered However, tracings in normal individuals were more difficult to obtain until recently

HISTORY

The hepatic tracing was studied first by Potain[10] then by MacKenzie[8] It was soon recognized that this record is particularly important because the liver reflects changes of flow and pressure of the inferior cava without distortion by an underlying artery On the other hand pulsations of the heart, transmitted through the diaphragm are possible and may be superimposed on the liver pulsations

The first stage involved the use of a large, round collector pressed against the skin of the right hypochondrium and of a Marey tambour[3, 8, 10, 11]

A second stage was based on the substitution of the Frank s or the Wiggers Dean s capsule for the tambour,[5] a greater accuracy of the tracing was obtained through optical magnification and photographic recording The latest development due to the author,[6, 7] consists of the use of a linear microphone and a galvanometer

TECHNIC

Modern procedure is based on the fact that a crystal microphone of a linear type[9] transforms pulsations of the air contained in the applicator into electrical waves These are recorded by the galvanometer of an electrocardiograph and are transcribed photographically or by direct writing methods An other sound or mechanical tracing should be simultaneously recorded in order to time the waves accurately Whenever the waves are small and magnification is necessary a preamplifier may be introduced between the crystal microphone and the galvanometer

The sensitivity of the linear microphone is such that the round bell normally used for taking low frequency tracings of the chest is sufficient for recording hepatic tracings A microphone connected to the cup as a support is placed over the right upper quadrant of the abdomen and is held by a rubber strap If respiration is forceful and irregular the microphone may be held by hand However this procedure is not recommended because slight movements of the hand may be recorded by the microphone In normal individuals the same procedure may be used on that part of the liver which crosses the epigastrium by firmly applying the cup below the right costal arch (Fig 54)

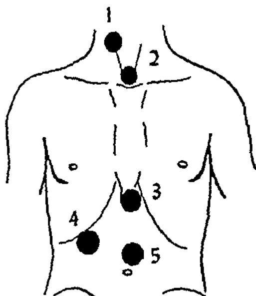


Fig 54 Location of the funnel for recording special tracings *1* carotid tracing *2* suprasternal (aortic) tracing *3* epigastric tracing, *4* hepatic tracing *5* abdominal (aortic) tracing

Respiratory movements possess an amplitude far greater than the hepatic waves therefore it is necessary for the patient to 'hold his breath for a few seconds during the taking of the tracing If the patient is unable to do so closing of the nose and mouth by the hand for a few seconds may be necessary Otherwise a high pass electric filter may be used This reduces the amplitude of all slow waves so that the shape of certain hepatic waves is somewhat changed Detection of a plateau might be more difficult with the use of the filter

ANALYSIS OF WAVES

The hepatic tracing shows a small positive wave during presystole ("a wave) It is due to presystolic swelling of the liver when atrial contraction arrests the venous flow Regurgitation of blood in presystole is minimal in normal individuals while it may be important in subjects with high venous pressure

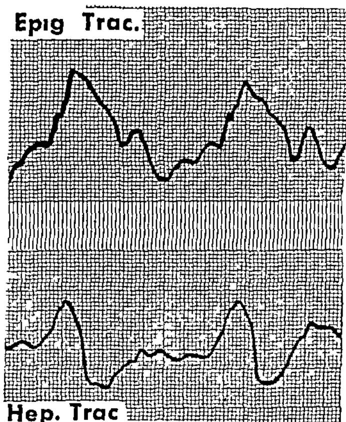


FIG 55 Hepatic (*Hep*) and epigastric (*Epig*) tracings simultaneously recorded in a normal subject In this case the hepatic tracing has a marked early systolic impact

The (a) wave of the jugular tracing and that of the hepatic tracing are practically simultaneous However the jugular wave may precede the hepatic wave especially if the former is recorded through an amplifier (Fig 57) The rise of the (a) wave of the hepatic tracing precedes the first sound at the apex by about 0 15 second Tracings of patients with high venous pressure and distended liver may present an atrial wave which is much higher in the hepatic than in the epigastric tracing proving the hepatic origin of this wave

In exceptional cases, a double presystolic wave may be recorded The first (a) is *transmitted through the diaphragm* via the epigastrium, the second (a) is a real *hepatic wave*

The beginning of ventricular contraction is revealed by *a small positive wave* (wave 1) probably transmitted through the diaphragm

Dressler [3] confirming fluoroscopic observations of Hitzenberger,[4] described a systolic drop of the right diaphragm (decreased volume of the liver) *in contrast* with a systolic rise of the left diaphragm (suction created by decreased intrathoracic pressure) This is confirmed by the fact that during most of systole the normal hepatic tracing shows a deep negative wave the *systolic collapse* In normal individuals the systolic collapse recorded below the right costal arch is far deeper than that recorded below the left costal arch (*Fig 56*) Therefore the former is the result of decreased hepatic volume while the latter is only due to rise of the diaphragm *with decrease of abdominal* pressure Sometimes a small notch can be seen during that *part of the first sound* which coincides with the

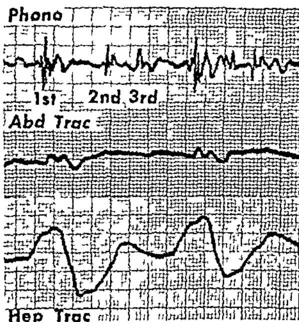


Fig 56 Phonocardiogram abdominal tracing and hepatic tracing *in a normal subject* (*The abdominal tracing was recorded from an area diametrically opposed to that of the hepatic tracing*)

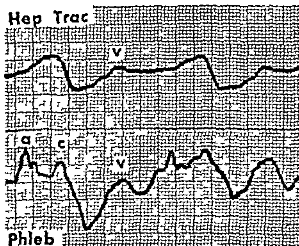


Fig 57 Phonocardiogram hepatic tracing and jugular tracing (amplified) in a normal subject

opening of the semilunar valves and during the wave 1b of the cardiogram, it is probably transmitted through the diaphragm

The tracing rises gradually at first rapidly later, with a peak (v) at the time of the opening of the tricuspid valve Following this, the curve falls forming a deep and rounded negative wave variable in shape and depth because of the effect of respiration of the venous return The lowest point of this wave coincides with the wave of rapid filling in the cardiogram and should be named 3 It may also be called *diastolic collapse*

The normal hepatic tracing is mainly formed by a deep negative wave, the systolic collapse, which is preceded by a positive atrial wave and is followed by a diastolic collapse (Figs 55, 56 and 57) These waves reflect the volume changes of the liver due to changes of flow in the inferior vena cava Smaller notches are transmitted from the heart through the diaphragm Transmitted right heart movements and aortic pulsations may be overlooked in normal subjects However it is wise to always compare the hepatic tracing with the epigastric tracing in cardiac patients

It should be kept in mind that a right ventricular impact is transcribed as an early systolic wave followed by a deep depression while the positive hepatic pulse of tricuspid insufficiency consists of a positive systolic plateau (p 356)

CONCLUSIONS

The hepatic tracing may yield important data in cases of diseases of the tricuspid valve, in heart failure and in adhesive pericarditis The hepatic tracing is superior to the jugular tracing because it is not influenced by an underlying artery, transmitting systolic pulses

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SECTION D

Intrathoracic Tracings

CHAPTER 10

Tracings of the Pulsations of the Esophagus

(ESOPHAGOCARDIOGRAPHY)

HISTORY

Tracings from the esophagus of dogs and humans were first recorded by Fredericq in 1886[3] The same author later identified most of the waves recorded with those presented by the atrial pressure curve[4] Early studies of the esophagocardiogram include those of Sarolea[15] Luciani[8] Minkowski[11] Young and Hewlett[18] Rautenberg[14] Lian[7] Janowski[6] Clerc and Esmein and Pace[12]

Later studies with different technics were made by Weitz and Schall[17] Boeckelmann[1] Taquini[16] Puddu[13] Luisada[9, 10] and Groedel and Miller The basic studies are those of Luciani, Rautenberg Boeckelmann and Taquini

All methods are based on the introduction of a tube into the esophagus The earlier group of workers connected the tube with a Marey tambour A second method made use of a Frank s capsule and of photographic recording[1, 13, 16] In recent years the tube has been connected with a crystal microphone of the linear type[5, 9, 10] or with an electromanometer[19]

TECHNIC

The best type of stomach tube is one of 5 mm internal bore This may be connected with a rubber cylinder having a metal support or a small balloon slightly distended by air * The tube should be marked in centimeters and should be radio opaque so that its descent may be followed by fluoroscopy It should be kept in mind that the esophagus curves in its lower section and proceeds from the vicinity of the spine into the anterior mediastinum below the heart According to the position of the tip four types of curves can be obtained (Fig 58)

1 At the aortic level 25–30 cm from the dental arch
2 At a high atrial level 30–35 cm from the dental arch
3 At a low atrial level 35–40 cm from the dental arch
4 At the ventricular level 40–45 cm from the dental arch

Each of the tracings presents interest However the second and third positions are the most important

ANALYSIS OF WAVES

Tracing at the Aortic Level

The following characteristics may be observed A small negative wave during presystole 0 05–0 08 after the beginning of P in the electrocardio gram (Wave 4) A small diphasic wave at the beginning of systole with in scription of the first large vibration of the first sound (Wave 1a) A deep sys tolic collapse during the ejection period interrupted by a small positive wave simultaneous with the (c) wave of the jugular tracing A rapid rise coinciding with the second sound and the incisura of the carotid pulse (Wave 2a) A rapid rise with a positive peak at the time of the third sound (Wave 3)

Tracing at the High Atrial Level

Usually a negative wave is present during presystole (Wave 4) A positive peak is simultaneous with the first sound (Wave 1) Then there is a small dip and a small peak synchronous with the (c) wave of the jugular tracing Later a rounded negative wave occurs during ejection and culminates with a positive peak at the time of the second sound (Wave 2) Last a negative peak is present during rapid filling of the ventricles (Wave 3) (Fig 58)

Tracing at the Low Atrial Level (True Atrial Tracing)

There is a negative wave during presystole with a peak which follows by 0 03–0 04 that of the P wave of the electrocardiogram (Wave 4) This

* A thin (0 1 mm) rubber membrane tied around the tip of a stomach tube may be used for the purpose

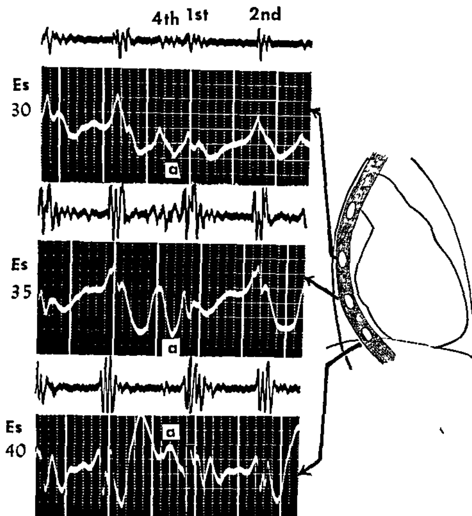


FIG 58 Esophagocardiograms and esophageal phonocardiograms at three different levels in a normal young man *Top* at 30 cm from the dental arch (high atrial level) *Middle*, at 35 cm from the dental arch (low atrial level) *Bottom* At 40 cm from the dental arch (high ventricular level)

wave may be double peaked (Fig 59) After this and starting after R a small positive wave (Wave 1a) occurs during the first part of the first sound This is followed by a small drop and another small positive wave synchronous with the second part of the first heart sound (Wave 1b) Then there is a systolic collapse and a positive or negative peak at the time of the second sound (Wave 2) Rapid ventricular filling is marked by a deep collapse (Wave 3) (Figs 58, 59)

Tracing at the Ventricular Level

The presystolic wave (4) is more frequently positive than negative The systolic collapse is poorly visible There are large vibrations caused by the

valvular events and simultaneous with the heart sounds (1a 1b 2a) Rapid filling is marked by a high positive wave (3) (Fig 58)

INTERPRETATION

Several factors act on the intraesophageal pressure and give rise to the waves of the esophagocardiogram

1 *Variations of Intrathoracic Pressure* These will be analyzed in Chapter 11 One of the main effects is the deep systolic collapse of the tracing at the aortic level

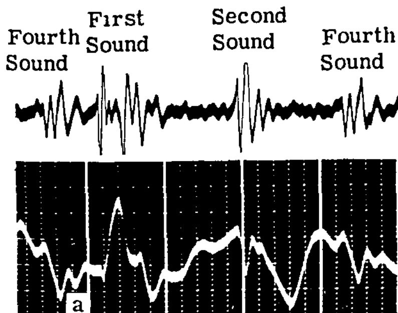


Fig 59 Esophagocardiogram and esophageal phonocardiogram (stethoscopic) at 35 cm from the dental arch in a normal adult Double atrial wave (*a*) in the mechanical tracing multiple atrial vibrations (fourth sound) in the sound tracing

2 *Changes in Volume of the Left Atrium* This chamber is in the near proximity of the esophagus Its contraction which takes place in presystole causes a negative wave in the tracing at the low atrial level and sometimes also in that at the high atrial level Lowering of the atrioventricular floor by the left ventricle causes the systolic collapse at the low atrial level

3 *Pulsations of the Descending Aorta* These may be observed in the tracing recorded at the high atrial level as a positive systolic wave

4 *Impact of the Ventricular Contraction* This can be observed in most tracings chiefly during the tension period

5 *Vibrations of the Heart Sounds* These are inscribed in most of the tracings

CONCLUSIONS

Until a few years ago, the esophagocardiogram had a unique position This tracing recorded at the atrial level permitted to obtain direct evidence of the movements of the left atrium Its interest was particularly marked in patients with a systolic murmur where a differential diagnosis between rheumatic mitral disease and other cardiac diseases was necessary At present, electrokymography represents an easier and better tolerated method of study Still esophagocardiography presents some interest In particular whenever a fluoroscope and an electrokymograph are not available the esophagocardiogram may be the method of choice in doubtful cases especially by using the electromanometer

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CHAPTER 11

Tracings of the Pulsations of the Air Passages

(INTERNAL PNEUMOCARDIOGRAPHY)

The term internal pneumocardiogram is now applied to the tracing of pulsations of the air passages and lungs as a consequence of the heart beat Previous terms were negative thoracic pulse cardiopneumatic waves 'respiratory pulse and 'heart notchings of the respiratory curve

HISTORY

Older studies showed that the heart beat causes changes of pressure within the thorax and movements of air through the respiratory passages which usually are not accompanied by audible sounds In heart diseases these movements may increase in such a way that the observer becomes aware of them, and there may be a subjective sensation

The first clinical observations were made between 1867 and 1888 by Friedreich[11] Galvagni[13, 14] and Cheesmann[5] more recently Gerini[15] Fischer,[7] Binetti and Lang[3] published clinical studies Tracheal or oral murmurs with a cardiac rhythm were described in aortic insufficiency and aneurysms of the aorta The name oral whiff' was used by Cheesmann[5]

Pressure Curves

Buisson[4] studied the so called negative thoracic pulse in animals and explained it with the decrease in volume of the heart as a result of ventricular systole Bert[1] studied the pulsations of air of the trachea due to the movements of the heart Landois [7] obtained records of air pulsations from the mouths of normal subjects He described negative waves when the glottis was open and positive waves when it was closed The latter were due to pulsation of the oral vessels Mosso[35] recorded the same tracing and described a double notched wave due to systolic diminution of pressure within the chest He attributed the first notch to expansion of the chest caused by the apex beat the second to diminution of cardiac volume However his records reproduced by Luciani [30] are not above reproach Klemensiewicz [5] and Haykraft and Edie[17] concluded that the pulsations were mainly due to rhythmic compression of the lung by the heart apex François Frank[10] attributed the waves recorded when the chest was open to pulsations of the pulmonary vessels Clinical pneumocardiograms were recorded by Siciliano [40] Cremer and Matthes [6] and Frugoni [7] in individual cases

In 1918 Klewitz [6] described the pneumocardiogram in detail He noted a small negative wave during presystole and a larger one during systole Small notches coincided with the two heart sounds

Pneumotachograms

A third phase started with the description of the pneumotachograph This apparatus records a tracing of velocity of the air flow during normal respiration[3, 8] (p 215) The cardiac notches of the pneumotachogram were studied by the author[31] in normal subjects and cardiac patients and were compared with those of the pressure curve They were found basically identical Actual backflow of air was found only during apnea Three main negative waves were described one presystolic one systolic and one diastolic A high positive pulse sometimes occurred in untrained patients when asked to hold their breath because they closed their glottis

Hochrein and Weiss[21] studied by means of a pneumotachograph the pulsations of the air caused by the heart beat during inspiratory and expiratory standstill No definite notchings were observed in normal subjects on the other hand positive waves were found in both phases of apnea in aortic aneurysm only during inspiratory apnea in adhesive pericarditis The study was repeated later by Hochrein[19] and by Hochrein and Laplace [0] Both the technic and the conclusions of these studies were discussed by Fleisch [9] Holzloehner [2] Hitzenberger and Hinteregger [18] Luisada [32, 33, 34] and Rubino [39] Later studies by Holzloehner[3, 4] were mostly based on the use of a pneumotachograph and a string anemometer

The most recent technic was described by the author,[35] who used a crystal microphone a high pass filter, and a galvanometer Subsequent studies were made by Groedel [16]

TECHNIC

A Sanborn Stetho Cardiette or Twin Beam Cardiette is used for the simul taneous *registration of the pneumocardiogram and the phonocardiogram* The latter is useful for timing the waves of the former A crystal microphone of the linear type[31] is used in conjunction with a high pass filter which de creases the slow respiratory deflections of the baseline without curtailing the rapid pulsations caused by the heart The jack of the linear microphone is inserted into the filter, that of the filter into the electrocardiograph The filter can be varied in the degree of attenuation of the slow respiratory deflec tions If three simultaneous registrations are needed a Tri Beam Stetho Cardiette or a Poly Viso with direct writing can be used

A 10 inch rubber tube connected to the microphone ends in an olive of bakelite inserted into one of the nostrils The patient is placed in a comfortable sitting or semirecumbent position with complete muscular relaxation and is instructed to breathe through the nose with his mouth closed

Two operating adjustments can be made the degree of filter attenuation upon slow respiratory waves and the amplification of the electrocardiograph Regulation of both gives a tracing having waves from 1 to 2 cm high while even the extreme phases of respiration are recorded The pneumocardiogram is registered at a film speed of 50 to 75 mm per second

If the patient is able to breathe evenly and slowly the filter may be dispensed with and the accuracy of the tracing is increased

Calibration of the pneumocardiographic waves can be done by using a device which produces a known change of pressure in the tube connected to the patient s nostril The pneumocardiographic deflections may then be ex pressed in millimeters of water

The electrical filter does not remove completely the low frequency com ponents caused by respiration Thus some waves may be seen in one cycle and not in a succeeding one because of the unlike rhythms of respiration and cardiac action These respiratory components are more marked in children and excitable adults Pneumocardiographic waves may be differentiated from respiratory waves because they occur in every succeeding cardiac cycle, al though somewhat modified in contour

ANALYSIS OF WAVES

It has been proven that the waves of the internal pneumocardiogram mirror the changes of intrathoracic pressure Apart from respiratory actions,

the latter are affected chiefly by the balance between flows of blood from and into the thorax Rapid outflow not compensated by inflow, or arrest of inflow while the outflow continues cause a suction effect which is compensated by inflow of air and is revealed by a negative wave in the pressure tracing

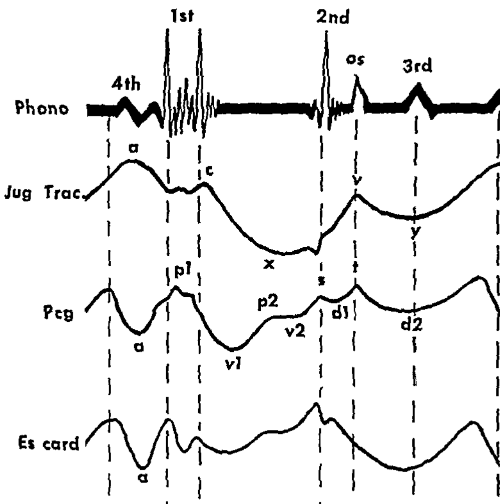


Fig 60 Comparison of the internal pneumocardiogram (*Pcg*) with the jugular tracing (*Jug*) and the esophagocardiogram (*Es card*) at the atrial level

Acceleration of inflow of blood into the chest without equivalent increase of outflow causes a pressure effect which is compensated by outflow of air and is revealed by a positive wave in the pressure tracing

The movements of two valves the tricuspid valve on the one hand and the aortic valve on the other are the most important because they regulate venous flow into and arterial flow from the chest

The normal pneumocardiogram presents *five negative waves* each connected with certain phases of cardiac action, and several small positive connecting points which may become *positive waves* (Figs 60 61, 62, and 63)

During presystole, a negative wave is present in the pneumocardiogram It is due to arrest of venous flow (or backflow into the large veins) caused by the contraction of the right atrium and should be called *Wave a* This results in a suction of air into the chest (Fig 63)

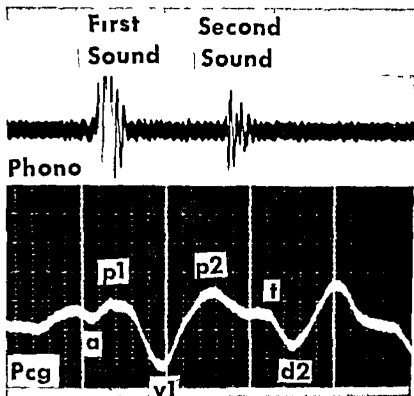


FIG 61 Phonocardiogram and internal pneumocardiogram of a normal young man (sitting relaxed filter)

A small positive wave occurs before the jugular tracing reaches the bottom between the a and c waves Lowering of the tricuspid leaflets by the papillary muscles creates a slight aspiration of blood causing outflow of air from the chest This notch is called p^1 (papillary contraction first positive wave)

During the first half of systole the blood leaves the thorax through the branches of the aortic arch and the abdominal aorta At the same time the venous blood either moves slowly toward the right atrium (inferior cava), or has a short backflow (superior cava or c wave of the jugular tracing) In this phase considerable aspiration of air into the thorax takes place and an im

portant depression is present in the pneumocardiogram This wave is often the most marked of all and is called v^1 (first ventricular wave)

After the first part of systole pronounced aspiration is exerted on the veins by increased negative pressure of the thorax and lowering of the floor of the right atrium This causes acceleration of the venous flow from both cavae as shown by the jugular and hepatic tracings The pneumocardiogram shows an upright notch which normally does not reach the zero line but in some cases may become a positive wave The pulsation of the pulmonary arteries* and

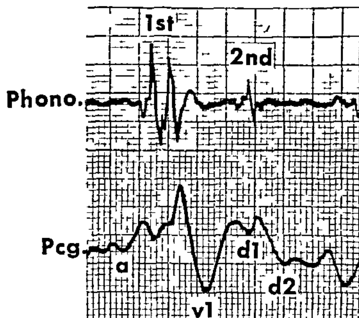


FIG 62 Phonocardiogram and internal pneumocardiogram of a normal young man (lying supine amplifier no filter)

that of the tracheal and nasopharyngeal vessels may contribute to the formation of this wave This wave is called *p* (peripheral pulse second positive wave)

During the second half of systole venous flow is slow because the right atrium is nearly filled while arterial flow from the aortic arch continues This causes suction of air into the chest and a negative wave This wave called v (second ventricular wave) may be deeper than v^1

* Theoretically the pulsation of the pulmonary vessels should not produce changes of the pneumocardiogram because the movement of blood is intrathoracic However the contact between small arteries of the lungs and alveolar air is so intimate that the decreased volume of the right ventricle affects more the venous flow while the increased volume of the lungs has a greater effect on the flow of air

An upright notch is simultaneous with the second sound and the incisura of the carotid tracing This is called *s* (semilunar valve closure) During the isometric relaxation period a straight line or a small downward wave is present The latter is called d^1 (first diastolic wave) Opening of the tricuspid valve causes a sudden onrush of blood from the venoatrial reservoir into the right ventricle and accelerates the venous flow The moment of opening is marked by an upright notch called *t* (tricuspid opening) It occurs simultaneously with the lowest point of the apex cardiogram (point 2b) and coincides with or slightly precedes the v wave of the jugular tracing

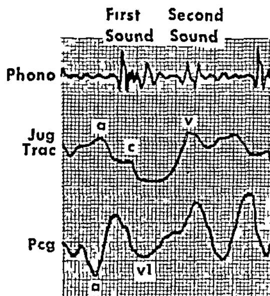


FIG 63 Phonocardiogram jugular tracing and internal pneumocardiogram of a normal young man (lying supine amplified no filter)

Blood leaves the thorax during the dicrotic wave forced by the elastic retraction of the aorta Its amount is larger than that of the blood entering the right atrium Therefore a suction effect is created and a negative wave d^2 occurs (second diastolic wave) This coincides approximately with the wave of rapid filling of the apex cardiogram and with the third sound

Additional waves may occur when diastole is prolonged as the result of variations of pressure within the thorax They are called d^3 d^4 etc (late diastolic waves)

PHYSIOLOGIC VARIATIONS OF THE PNEUMOCARDIOGRAM

Respiratory Changes

Normal subjects show deeper waves during the first part of inspiration However two waves may combine in a single one The *a* wave is deep and broad p^2 is smaller and of a short duration v^1 and v^2 tend to fuse into a single negative systolic wave Normal subjects present smaller waves during expiration especially in the second half of the phase Wave p^1 is usually clearly defined and sharp p^2 is tall broad and definitely positive, *t* is low *s* is taller and often well defined (Fig 64)

Inspiratory and Expiratory Standstills

The use of a crystal microphone and high pass filter eliminates the necessity of the patient holding his breath in extreme positions and avoids individual

variations and abnormal pressure conditions However, the author[35] compared these extreme phases with the technic of normal respiration The degree of inspiration or expiration is a variable depending upon training and cooperation of the patient, and greater or lesser effect upon the waves was found No fundamental difference in timing or polarity of the waves was found between the changes produced by normal respiratory phases and those resulting from *apnea in the extreme positions*

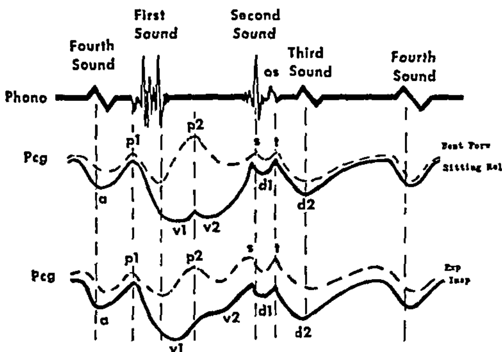


FIG 64 Respiratory and positional changes of the pneumocardiogram first *with subject bent forward and sitting relaxed and second during expiration and inspiration*

Changes Due to Position

The best tracings are obtained in either a sitting or a semirecumbent position with the muscles completely relaxed Other positions such as supine sitting erect sitting bent forward or standing may produce important changes *in the* internal pneumocardiogram (Fig 64) Many of these changes were studied by Holzloehner[23, 4] and explained as the result of an altered venous flow caused by modified tension of the venous walls

Bradycardia and Tachycardia

Because of short diastole subjects with tachycardia often show partial or total fusion of d^2 with *a* and small amplitude of d^1 These variations are ex

plained by changes in diastolic venous inflow and abrupt systolic collapse of the veins In bradycardia the waves are typical, and additional waves may occur during diastole

CONCLUSIONS

The pneumocardiogram presents a time relationship between four waves and four phases of the cardiac cycle

p^1 = closure of the tricuspid valve = first part of first sound

s = closure of the aortic valve = main wave of second sound

t = opening of the tricuspid valve = opening sound of tricuspid (and mitral)

d^2 = rapid inflow into the right ventricle = third sound

The internal pneumocardiogram has therefore some value as a timer of sounds recorded by the phonocardiogram The tracing presents interest whenever lesions of the tricuspid valve are suspected in adhesive and constrictive pericarditis, *and in syphilitic heart disease with aneurysm of the aorta or aortic* insufficiency

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CHAPTER 12

Tracings of the Pulsations from the Pleural Cavity

(PLEURAL TRACINGS)

The study of these pulsations has a limited value and had so far, few applications, largely because of the technic

Following studies of Rubino[2] in the author s laboratory, Borchardt and Groedel[1] studied by graphic methods the intrathoracic pressure changes in subjects with pneumothorax Complete identity between pneumocardiogram and intrathoracic pressure curves has been found

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SECTION E

Arterial Pulsations

The study of the arterial pulse has had a large part in the physical examination of a patient since the oldest times Chinese physicians described in detail this study before 2500 B C They examined the pulse of several arteries with light medium and strong compression of the vessel and they reckoned the pulse respiration ratio [5] Hippocrates gave details about rate amplitude strength and rhythm of the pulse as well as about the rapidity of its rise (*pulsus celer* vs *pulsus tardus*) Galenus added to these elements the tension and filling of the artery Weitbrecht[14] observed in 1734 that the carotid pulse preceded the radial His observation was purely clinical and was confirmed by graphic methods only one century later by Weber [13]

The beginning of graphic studies led to recording of three types of arteriograms the aortogram the carotid sphygmogram and the radial sphygmogram Later on the sphygmograms of several arteries have been recorded systematically *

* See References page 131

CHAPTER 13

Tracings of the Pulsations of the Aorta

(AORTOGRAPHY)

HISTORY

Aortograms were recorded in the horse by Chauveau and Marey[3] in 1863 and shortly afterwards by Fick[6] and Fredericq[7] in the dog Frank[1] used an optical system for recording aortograms in animals and his studies were followed by a score of others (v Frey, Huerthle Bayliss and Starling Tigerstedt)

The first tracings in man were recorded by Mueller and Weiss[1] Clinical cases of aortitis or aortic aneurysm were studied later by graphic methods (Cardarelli Zagari[10]) The author made systematic studies in normal subjects and cardiac patients[9, 10]

The tracing was recorded first with a funnel connected to a Marey tambour then with a Frank s capsule later with a crystal microphone

TECHNIC

The aortogram may be easily recorded over three points the *second right interspace* the *suprasternal notch* and the *abdomen* (Fig 65) The first record already described (p 70) is influenced by waves due to direct cardiac

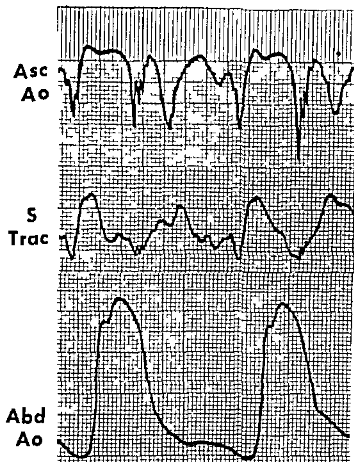


FIG 65 Simultaneous aortograms in a young woman *Upper* ascending aorta over the second right interspace (*Asc Ao*) *Middle* aortic arch at the suprasternal notch (*S Trac*) *Lower* abdominal aorta above the umbilicus (*Abd Ao*)

motion The second type of aortogram has less interference from cardiac waves Whenever the aortic arch is dilated or blood pressure is increased excellent tracings are obtained in this area and a pure arterial record can be observed The abdominal aortogram is recorded slightly above the umbilicus poor results are obtained when the abdomen is distended or obese while a good tracing is obtained in lean individuals

In all three types of aortogram a funnel connected to a linear microphone is placed against the skin over the vessel In the case of the suprasternal notch the funnel is held by hand or a suction cup is used In the other two instances a rubber band holds the funnel The simplest way is to use a stetho

scopic microphone as a support for the cup Thus simultaneous tracings of the aortic sounds and of the aortic pulse are recorded

The linear microphone[11] transforms pulsations of air into equivalent electrical pulsations so that the tracing can be recorded by a galvanometer of an electrocardiograph either on photographic film or by direct writing

ANALYSIS OF WAVES

The aortogram is essentially a tracing of central pulse However, tracings recorded in normal subjects at the second right interspace or at the suprasternal notch present additional waves due to changes of cardiac volume or simultaneous with the heart sounds (Fig 65)

An atrial wave in presystole (Wave 4) a small wave during the tension period (Wave 1a), and a wave of rapid filling in early diastole (Wave 3) are frequently observed The tracing shows a steep rise at the beginning of ejection and frequently presents a small depression in the ascending limb of the pulse (anacrotism) This is followed by a slower rise a rounded peak and a slow decline The end of ventricular contraction and the onset of ventricular relaxation are marked by a sudden drop of the tracing (incisura) After this, one or more coarse vibrations take place following the rebound of the blood on the closed aortic valve then a slow decline of the tracing can be seen

As soon as the ascending aorta and the aortic arch are dilated by the pulse the blood wave moves rapidly into the abdominal aorta and reaches the arteries of the legs The movement of blood during systole is complicated by the low natural frequency of the aortic wall and by reflected waves from regions with sudden narrowings and branchings[15] Experiments of Hamilton and Dow[8] have proven that the pulse wave presents progressive changes so that it gradually acquires a higher peak and a simpler contour This is due to the existence of a standing wave created by alternate accelerations and decelerations The arch and the upper part of the descending aorta accommodate more blood during the first part of ejection the lower aorta and its branches more blood during the second half[15] For this reason, the abdominal aortogram has a more pointed peak and shows less well the various details of the tracing Aortic catheterization in man has shown the following progressive changes[17]

1 The onset of the anacrotic limb occurs later and the anacrotic notch disappears
2 The incisura is gradually lost and replaced by a dip ending in the dicrotic notch Both this and the dicrotic wave become more pronounced toward the periphery

CONCLUSIONS

The aortogram is of use in the diagnosis of the following conditions diffuse enlargement (aortitis) or local dilatations (aneurysms) of the ascending aorta and aortic arch due to syphilitic heart disease (second space and suprasternal notch) aortic stenosis (second space and suprasternal notch), coarctation of the aorta (abdominal aorta), arteriosclerotic dilatation of the abdominal aorta (abdominal aorta), and differential diagnosis between aneurysms and tumors or masses having a transmitted pulsation

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CHAPTER 14

Tracings of the Central Pulse

(CAROTID AND SUBCLAVIAN SPHYGMOGRAPHY)

In addition to the aortogram a typical ' central pulse may be recorded over the carotid and subclavian arteries

HISTORY

Huerthle[4] was probably the first to record carotid tracings in man He used a Marey tambour and a specially built support fastened to the patient s head A similar device was used shortly afterwards by Edgren MacKenzie[5] used this method of investigation systematically Frank[7] recorded carotid tracings optically by connecting a funnel with his segment capsule Since then numer ous important studies have appeared Among them those of Wiggers[7] Wezler[6] and Hamilton[3] are outstanding The author has used a crystal microphone for recording these tracings since 1940

TECHNIC

The tracing of the carotid artery is obtained by pressing a small funnel *against the skin medial to the right sternocleidomastoideus* The pulse of the subclavian artery is obtained by placing the funnel over the medial third of the supraclavicular fossa The funnel may be held steadily by hand or may be

fastened and held by a special device like that supplied by the Cambridge Instrument Co of New York A third method consists of wrapping around the neck the double cuff of a device for recording pulse and blood pressure (p 154) The cuff is inflated at 15–20 mm Hg in order not to embarrass cerebral circulation The tracing obtained by this method presents strong venous components Still it may be useful whenever the patient needs to move his arms and neck, as in taking simultaneous tracings of electrokymography and sphygmography (p 178)

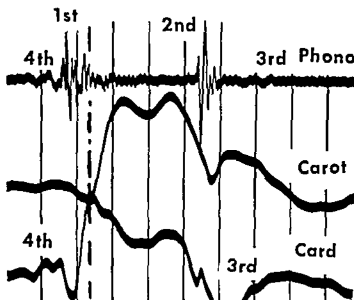


FIG 66 Phonocardiogram carotid tracing and apex cardiogram in a normal young man

ANALYSIS OF WAVES

The tracing of the central pulse reveals three main waves [1] the *percussion wave* the *tidal wave* and the *dicrotic wave* (Figs 31 38 and 66) The details of the tracing are as follows

1 One or two small waves are present during presystole and during the tension period of the ventricles

2 There is a rapidly ascending phase (the anacrotic slope) which frequently presents a change of speed (anacrotic depression) The rise of the curve coincides with that large vibration of the first sound complex which is caused by opening of the aortic valves (p 44)

3 The peak or summit of the percussion wave is attained at about the middle of systole and is followed by a slight depression

4 A second, more rounded wave (tidal wave), occurs during the second part of systole and is followed by the beginning of the descending phase (catacrotic slope)

5 In coincidence with the main vibration of the second sound complex (closure of the aortic valve), the curve presents a sudden drop (incisura), often followed by one or two small vibrations

6 The curve rises again forming a slow positive wave, the dicrotic wave

7 Later, the tracing gradually falls to its lowest level which is attained just prior to the rise of the following pulse wave

Reflected waves from peripheral subdivisions may be superimposed on the curve if this is taken at a high level of the carotid artery (upper carotid pulse) [7] the changes are noticeable during early ejection soon after the incisura They occur earlier if there is hypertonus or sclerosis of the wall, so that the speed of the pulse wave is increased Low tonus of the wall is revealed by a high peaked tracing [7]

The small waves which precede the rise of the pulse curve are transmitted *from the heart The first is caused by atrial contraction, the second* by bulging of the aortic valve during the tension period They are present only in the central pulse and are damped later by the walls of the peripheral arteries The rapid, ascending phase of the pulse curve is due to the ejection of blood into the aorta The anacrotic change of slope is due to the fact that at first the inertia prevents a large displacement of blood while later a more rapid flow occurs [1] the tonic elastic reaction of the aorta may also contribute to it The formation of a summit when about half of the blood has left the left ventricle may be attributed to the fact that the volume of blood entering the aorta is then smaller than that leaving the aorta through the various branches [1] The tidal wave seems due to the summation of the still moving wave with multiple waves reflected from the periphery [1]

The aorta and the left ventricle are still a single chamber at the beginning of ventricular diastole Ventricular relaxation causes a sharp drop of pressure in the aorta which is quickly terminated by closure of the semilunar valves this causes the incisura When pressure eddies complete the closure of the aortic valve the retraction of the aortic wall forces blood toward the heart as well as toward the periphery This sets up a negative wave which follows the main percussion wave (the incisura) After closure of the aortic valve the blood column rebounds from its surface and sets up a second positive wave (dicrotic wave), which follows the main wave toward the periphery

CONCLUSIONS

The tracing of the central pulse is useful mainly for timing the waves of other tracings For example hepatic tracings or electrokymograms are fre

quently timed by means of a carotid tracing The tracing of the central pulse gives accurate information about the time of opening of the aortic valve (rise of the pulse) and its closure (incisura of the pulse) Therefore this tracing presents interest in bundle branch block Abnormal patterns are found in atherosclerosis of the aorta coarctation of the aorta, aortic stenosis and aortic insufficiency The tracing helps in the differential diagnosis of these conditions

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CHAPTER 15

Tracings of the Peripheral Pulse

(BRACHIAL RADIAL FEMORAL TIBIAL PEDAL AND DIGITAL SPHYGMOGRAPHY)

HISTORY

The earliest studies of sphygmography (Vierordt [16] Landois[8]) were made by using a loaded rubber bulb applied over the radial artery and were extremely inaccurate The first reliable instrument with mechanical registration was Marey s sphygmograph[9] for recording the radial pulse (Fig 1 p 4) Dudgeon [4] Jaquet [2] and Frank and Petter[6] used similar devices However another model with air transmission writing through a Marey tambour was somewhat superior

Tracings obtained through optical recording represent a second stage in development Czermak[2] pasted a small mirror over the skin in the immediate vicinity of an artery and recorded the motion of a beam of light reflected by it Frank[5] and Ohm[11] used mechanical optical systems However in the above systems the contact with the artery was not perfect and may have been a source of error For this reason a new device was studied a pneumatic cuff was wrapped around the limb, pressure was set in the cuff and the tracing was recorded optically while the artery was submitted to a known compression (turgosphygmography[10, 14]) The best model for many years was that of Pachon Boulitte [12]

A third stage is represented by the use of a differential crystal microphone connected with a galvanometer and a blood pressure cuff (Rappaport and Luisada[13])

TECHNIC

By using the device of Rappaport and Luisada[13]* (Fig 7 p 155) the pulse tracing can be recorded on any of the four limbs or any of their sections

The double cuff is carefully wrapped around the segment A pressure of 20 mm Hg is established in the lower recording cuff The switch of the box is moved from adjust to register and further pressure is established in the upper cuff The tracing of the pulse can be taken at any compression It is advisable to note the level of compression, as well as that of the blood pressure in the limb prior to taking the record

Having already connected the jack of the instrument with the outlet of an electrocardiograph the switch of the latter is moved to 1 (in a direct writing instrument this is not needed), and the record is taken at a film speed of 25, 50 or 75 mm per second Pulse recording is based on the use of a linear

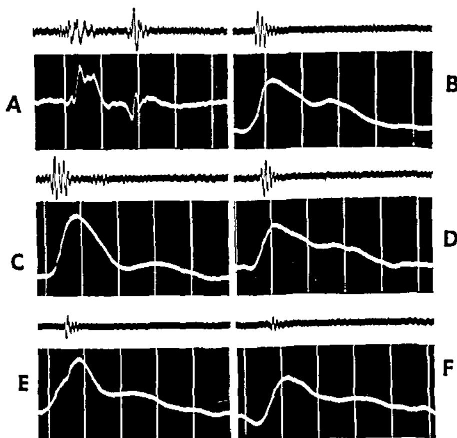


Fig 67 Sphygmograms and phonoarteriograms of a normal young man The sound tracing of *A* actually records the cardiac sounds *A* subclavian *B* brachial *C* femoral *D* radial *E* low tibial *F* pedal

* This is built upon special order by the Sanborn Co of Cambridge Massachusetts

microphone with two outlets Compression is developed on both sides of the crystal, so that this is not damaged When communication between the two is closed, the pulsations of the artery, transmitted to the cuff are received by one side of the crystal and transformed into electric pulsations The principle underlying the changes of contour of the pulse when pressure is increased in the upper cuff will be described later (p 155)

A more peripheral sphygmogram can be obtained by the use of a finger plethysmograph of mechanical or electrical type (p 148)

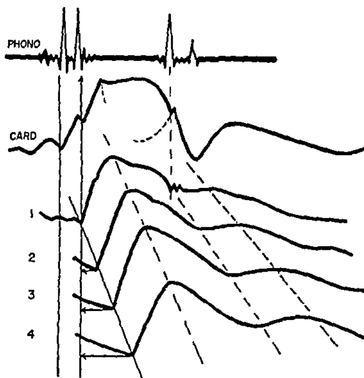


FIG 68 Modifications of the details of the sphygmogram from the central to the peripheral arteries *Phono* sound tracing of the heart *Card*, low frequency tracing of the chest wall (apex cardiogram), *1* subclavian pulse *2* brachial pulse, *3* radial pulse, *4* tibial pulse

ANALYSIS OF THE WAVES

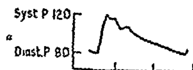
The peripheral pulse of normal subjects is much simpler than the central pulse The ascending phase is straight the incisura is replaced by a rounded depression The dicrotic wave is also rounded After the dicrotic wave, other secondary vibrations may occur (Fig 67)

The reaction of the peripheral arteries deepens the predicrotic notch transforming it into a valley, and rounds out the dicrotic wave Therefore the peripheral pulse shows no evidence of the tidal wave (Fig 68)

The abnormal sphygmogram may show marked variations

1 The *water hammer type* has a steep ascending slope and a rapid drop There is a turbulent condition at the front of the wave which may be revealed by a small anacrotic notch and is felt as a thrill upon palpation It is typical of aortic regurgitation

2 The *anacrotic pulse* has a notch in the ascending slope It is found in aortic stenosis hypertension and marked bradycardia



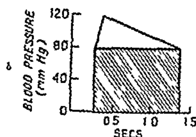


FIG 69 *a* Normal pulse tracing *b*, absolute sphygmogram (pressure time scheme for the same case) (From Bramwell and King's *Principles and Practice of Cardiology* Courtesy of the Oxford Univ Press)

3 The *dicrotic pulse* has a high dicrotic wave It is found in fever anemia and hyperthyroidism

4 *Pulsus alternans* is a regular pulse which has alternatively one smaller wave out of two It is found in cases with severe myocardial damage (p 428)

5 *Pulsus paradoxus* is a severe periodical waxing and waning of the pulse connected with respiration and is typical of constrictive pericarditis (p 461)

A measurement which seems to have a certain clinical value is that of *crest time* This is measured in seconds between the rise of the pulse and the highest point of the main wave[4, 15] Normal crest time of the radial pulse is between 10 and 16 per cent of the total duration of the pulse cycle from rise of one pulse to rise of the next (Fig 261)

THE ABSOLUTE SPHYGMOGRAM

The sphygmogram or pulse tracing is a pressure tracing of the artery However, it does not reveal the pressure which is between zero and the foot of the wave A diagram called the *absolute sphygmogram*, evaluates the various elements of the pulse In this diagram the ordinate represents pressure (measured by the sphygmomanometer) while the abscissa represents time and is based on the pulse rate (Fig 69)

APPLICATIONS

The sphygmogram can be used for several purposes

1 SPEED OF THE PULSE WAVE Simultaneous phonocardiograms (over the aortic area) and arterial tracings are recorded The distance between the second large vibration of the first sound (opening of the semilunar valves)

and the rise of the pulse gives the speed of the wave if compared with the time lines of the record The times between the opening of the semilunar valve and the arterial pulses of different arteries in a normal young man are reported in Table 5

TABLE 5 SPEED OF PULSE WAVE MEASUREMENTS IN NORMAL SUBJECTS

Artery	*Time in seconds from opening of semilunar valve to rise of the pulse*	*Time in seconds from closing of semilunar valve to incisura of the pulse*	*Time in seconds from second sound to peak of the dicrotic wave*
Subclavian	0 02	0 02	0 16
Abd aorta	0 08	0 08	0 20
Brachial	0 08	0 14	0 30
Femoral	0 10	0 16	0 32
Radial	0 12	0 16	0 35
Tibial	0 18	0 36	0 46

2 Shape of the Pulse Wave Records should be taken at diastolic pressure, at mean pressure and just below systolic pressure The last shows prominence of all secondary waves including the anacrotic notch Measure ment of crest time" is made in tracings recorded at diastolic pressure
3 Study of Arterial Sounds This sound tracing is automatically re corded with the sphygmogram by using the above described apparatus (p 143)
4 Irregularity of the Heart Simultaneous electrocardiograms and sphygmograms are taken The former gives information about the type of arrhythmia, the latter shows the peripheral effect of the disorder
5 Study of the Peripheral Circulation The study of the arterial pulse may be important in cases of arteritis thrombosis or embolism The pneumatic cuff is successively applied to different sections of the limb and the sphygmogram is recorded at the lowest pressure capable of giving a good record Crest time is measured in both the tracing of the medium sized arteries and in that of fingers or toes
6 Measure of Stroke Volume A method for calculating stroke volume on the basis of the study of the pulse was described by Wezler and Boeger[17] *

* The method is based on
1 Measurement of aortic section (*S*) by orthodiagraphy
2 Pulse pressure (*PP*) measurement by sphygmomanometry
3 Determination of the speed of the pulse (*A*) through comparison of carotid and femoral tracings
4 Determination of length of the pulse wave (*e*) from peak of the main wave to peak of the dicrotic wave
Stroke volume (*I*) is calculated by formula (*I*)

$$I = \frac{2\ PP}{E} \quad (1)$$

It was further studied by Altana,[1] Grishman and Master [7] and Schmid and Reubi [15] As the difference between the values found by this method may reach 50 per cent, the method is unreliable

CONCLUSIONS

Multiple studies concerning the function of the left ventricle the structural changes and the function of the peripheral arteries can be based on sphygmographic studies

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E is the coefficient of elasticity of the vascular segment from the aorta to the femoral artery and is calculated by formula (2)

$$E = \frac{1.06 \times a^2}{S \quad \frac{1}{4}} \qquad (2)$$

(1 06 is the specific gravity of the blood)

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CHAPTER 16

Sound Tracings of Peripheral Vessels

(PHONOARTERIOGRAPHY PHONOPHLEBOGRAPHY)

HISTORY

While the peripheral arteries of normal subjects present no sounds ausculta tion of the carotid and subclavian arteries reveals transmission of the heart sounds Compression of an artery causes the appearance of sounds or blowing murmurs a fact which has been used for recording blood pressure (Korotkow method) Arterial sounds may be present in abnormal conditions over periph eral arteries or veins

Sound tracings of peripheral arteries have been taken in order to obtain a graphic record of blood pressure[7, 8] or of special sound phenomena[1, 3, 4, 5, 6] Studies by Groedel and Miller[2] of the phonocardiogram of the neck probably include venous and arterial sounds of local origin

TECHNIC

The sound tracing from the arteries of the neck can be recorded by applying a microphone over the carotid or subclavian artery This is provided with a medium sized funnel and is held in place by a rubber strap

A sound tracing from the veins of the neck can be obtained by a suction cup (p 95) connected to a linear and a stethoscopic microphone Two simul taneous tracings of sounds and pulsations are thus obtained

A sound tracing from the arteries of the limbs can be obtained by using the apparatus of Rappaport and Luisada [7] The double cuff is wrapped around the limb The lower cuff is inflated to the desired pressure level after switching to "register (p 154) Simultaneous pulse and sound tracings are then obtained through a linear and a stethoscopic microphone

ANALYSIS OF THE WAVES

Two sounds are generally recorded over the arteries of the neck (Fig 67) The first of them occurs at the time of the rise of the pulse, the second at the

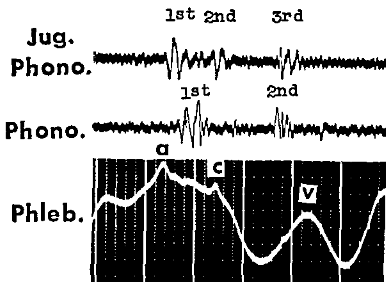


Fig 70 Jugular sounds *Jug Phono* sound tracing recorded over the right jugular vein *Phono*, phonocardiogram *Phleb* jugular tracing The first and second jugular sounds coincide with two waves (*a c*) of the vein while the third coincides with the second heart sound

time of the incisura The first sound has several vibrations and is frequently divided into two groups the first group slightly precedes the rise of the pulse and is due to transmission of the first part of the first heart sound, the second group coincides with the maximum dilatation of the vessel and is of local origin The second sound is probably transmitted from the heart (second heart sound)

One single sound is recorded over the arteries of the limbs if these are compressed The sound coincides with the distention of the artery by the pulse wave (Figs 67A, 76) More than one sound is recorded in certain clinical conditions (p 342)

The sounds recorded over the veins of the neck are usually three (Fig 70) The presystolic sound is the largest and is made of several vibrations It may

be due to transmission of the fourth sound of the heart through the upper mediastinum but distention of the vein in presystole is likely to be an important contributory factor

The sound coinciding with the c wave is probably of local origin The third is largely a transmission of the second heart sound

Normal peripheral arteries reveal only one sound e g a multiple vibration at the time of maximal dilatation This sound becomes weaker and simpler in the more distal vessels More than one sound can be recorded on the other hand in clinical conditions

CONCLUSIONS

Records of sounds and murmurs from the peripheral arteries and veins are not part of the regular study of a cardiac patient They may be used in individual cases either to obtain graphic evidence of unusual auscultatory phenomena or for research It should be kept in mind that occasionally, sounds and murmurs originated in the heart are *recorded best in the neck* especially in obese and emphysematous patients

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CHAPTER 17

Tracings of the Volume of the Limbs, of their Segments, or of the Abdomen

(PLETHYSMOGRAPHY)

A plethysmogram is a tracing of the volume changes of any part of the body on account of changes in its blood content In order to obtain a plethysmogram of a section of the body, this should be enclosed in a rigid container Inflow and outflow of blood should be unhampered by the membrane which insures closure of the container around the part of the body The most common plethysmograms are those of the limbs (forearm leg), or parts of them (fingers toes) A plethysmogram of the abdomen is possible in man A plethysmogram of the brain has been recorded after removal of part of the skull following injuries

HISTORY

Plethysmograms of the forearm or the leg were recorded in man by Buisson[3] Fick,[7] Mosso[14] and Mueller[15] between 1860 and 1900 and by Brodie and Russell[2] in 1905 (Fig 3) Abdominal plethysmography was studied first by Weber[18] in 1910 Cerebral plethysmography was studied in the same year in cases with skull injuries by Mueller and Veiel[16] Systematic studies in cardiac patients were made by Weber[19] in 1916 Interest in the method lagged for some

time The latest method is that of photoelectric plethysmography, described by Matthes[13] in 1935 and developed by Hertzman and coworkers[8 9 10]

TECHNIC

General Rules[17]

The container has rigid walls It is closed around the limb by means of a rubber membrane, carefully applied Water at body temperature fills the container and transmits volume changes to a recording apparatus This works with little change of tension at any level Air transmission has been used but is less exact Rapid undulations of the tracing are caused by arterial pulsations Slow movements of the curve are due to changes in the blood content usually connected with variations of vascular tonus

1 Fluid Displacement Method[1 0]

This method employs a water air system in which the enclosed extremity is surrounded by water and displacements of small volumes of air are recorded Systems employing only water are more accurate but have difficulties due to the hydrostatic pressure on the enclosed limb A *sectional plethysmograph* encloses only a segment of an extremity like the hand part of the forearm or of the leg or the foot

The chamber is a metal box with circular openings at each end through which the extremity is placed Individually fitted nonconstricting rubber cuffs of thin rubber sheets are cemented to the shaved skin of the limb at either end of the chamber The cuffs are fastened by heavy rubber bands and adjustable metal collars to projecting rims around each opening of the chamber and are supported by adjustable diaphragms to prevent distention

Both photographic and direct writing methods can be used for the records A small Krogh spirometer or a Brodie bellows connected with the air layer of the chamber is used for the transcription The system is calibrated by adding 1 cc of water by means of a Cornwall syringe Arterial inflow can be calculated by means of a simple formula [0]

A small portable plethysmograph* has been developed by Burch[4 5] The fingertip is enclosed within a small cup three quarters full of water changes in pressure of the air content above the latter are transmitted to a capsule which is connected to a short aluminum arm carrying a quartz string The motions of the string are recorded optically

Abdominal plethysmography[17] can be obtained in the following way A rubber tube closed by a membrane is introduced into the rectum The tube is filled with water at body temperature which distends the membrane like a

* This is built by The Cambridge Instrument Company of New York

bag and transmits pressure changes to a recording apparatus The abdomen is *then enclosed in a tight girdle, so that its wall becomes practically rigid* Changes of the tracing due to the respiratory movements of the diaphragm are revealed by regular and periodic oscillations of the basal line Changes caused by intestinal movements are easily recognized, being irregular and sometimes rapid Changes due to variations of the blood content are revealed, on the other hand, by slower and nonperiodic, easily recognizable, changes of the base line The applicability and the sensitivity of this method are remarkable

2 Pressure Method[11 12 20]

Pressure is exerted on the surface of the forearm to collapse the blood vessels before the volume of the *segment is determined By means of* appropriate calculations the number of cubic centimeters of fluid filtered or absorbed per 100 cc of forearm tissue can be determined The apparatus consists of a water filled metal cylinder with an inner rubber sleeve connected by pressure tubing to a 200 cc burette By opening a valve to an air pressure reservoir 200 mm Hg of pressure is applied to the water of the burette and transmitted to the plethysmograph thereby compressing the enclosed segment Readings are made at the end of two minutes of compression then pressure is released The volume recorded has been called "reduced arm volume

3 Photoelectric Method

This method has been developed by Hertzman and co workers[8 9 10] It is based on penetration of light through the tissues of a fingertip and recording of its variations by means of a phototube Recording devices vary from simple galvanometric arrangements to direct coupled amplifiers of special design A relatively simple arrangement has been used in the author s laboratory This includes the use of a modified Sanborn electrokymograph connected with an electrocardiograph An RCA 1P22 phototube, sensitive to red rays, is mounted in the pickup unit and placed facing the upper surface of a fingertip A flash light bulb directing its light upwards under the finger, and a set of dry batteries are used for illuminating the finger If the hand of the patient is covered with a black cloth, recording can be done with normal light in the room The switches of the electrokymograph and electrocardiograph are set for optimal amplification The record can be taken at speeds of 10 25 or 50 mm per *second*

Calibration of the deflection on the record in terms of changes in blood content is obtained by comparison with the deflection produced by the insertion of a glass plate (microscopic slide 1 1 mm thick) in front of the phototube On account of the various stages of amplification involved this set up can be used mainly for an exact study of the rapid arterial pulsations (Figs 71 and 72) Changes due to vasomotor reactions are chiefly related to *increased or decreased amplitude of pulsations* (Fig 73)

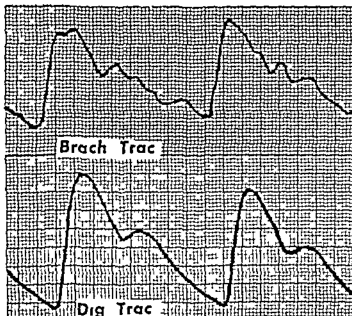


Fig 71 Photoelectric plethysmogram of the index finger (*Dig Trac*) compared with the brachial tracing of the other arm (*Brach Trac*) Delay of the digital pulse over the brachial pulse

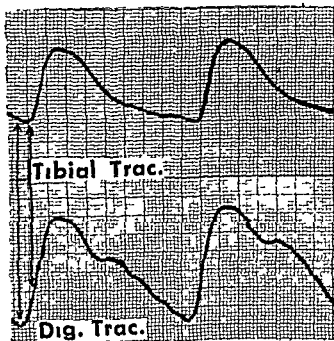


Fig 72 Photoelectric plethysmogram of the index finger (*Dig Trac*) compared with the tibial tracing (*Tibial Trac*) in a normal young man Delay of the tibial over the digital pulse

Separation of the arterial from the venous components of the plethysmogram can be attempted in the following way [0] A cuff is placed over the proximal part of the limb under study, and is rapidly inflated under a pressure of 30–40 mm Hg in order to block venous outflow Arterial inflow is then calculated from the slope of a line drawn through corresponding points on the first two or three pulse waves following venous occlusion The base line is obtained by connecting corresponding points on the pulse waves immediately preceding venous occlusion

ANALYSIS OF THE WAVES

The plethysmogram is a record of the changes in volume of the observed section in relation to time These changes may be due to fluctuation of three types of fluid blood, intracellular and intercellular fluid lymph In the average subject variations of intracellular and intercellular fluid are unlikely lymph volume changes may contribute only to slow deflections On the other hand variations in blood volume of the segment are responsible for most of the observed waves Five types of waves the faster superimposed on the slower have been observed [5]

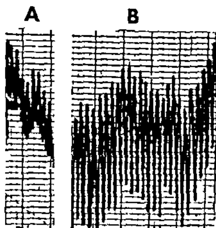


Fig 73 Photoelectric plethysmogram at low speed Before and after inhalation of amyl nitrite

1 *Pulse waves* caused by the contraction of the heart They represent the difference between arterial inflow and venous outflow at each cardiac beat They have a cardiac rhythm and their amplitude varies considerably following vasomotor changes They are smaller during vasoconstriction

2 *Respiratory deflections* which follow the respiratory rate and are due to respiratory fluctuations of both the arterial and the venous flow

3 *Alpha waves* which are present in all parts of the body, they vary in frequency and size and are by no means uniform in contour They are not related to variations of arterial pressure but seem to be due to local changes of vascular tonus because they disappear after sympathetic block or sympathectomy Their rate is slower than that of respiration and is about ten times per minute

4 *Beta waves* which are large slow and irregular waves occurring about one to two per minute

5 *Gamma waves* are the slowest of all including cycles of from one to eight hours They represent extensive changes in volume

The study of the pulse of the fingers and toes should be correlated with that of the radial and tibial pulses and their crest times (p 139 Fig 259) should be compared In normal subjects digital crest time is equal to or not longer than 4 per cent above radial crest time [6] The increased amplitude of the pulse after exertion is used as a functional test of the efficiency of the peripheral arteries (Fig 262)

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SECTION F

Pressure Tracings

CHAPTER 18

Tracings of Arterial or Venous Pressure

(GRAPHIC SPHYGMOMANOMETRY OR PHLEBOMANOMETRY)

HISTORY

In 1733 Stephen Hales[1] inserted a cannula into the femoral artery of a horse and allowed the blood to rise in a vertical tube the pressure was calculated from the height of the pulsating blood column In 1828 Poiseuille[37] substituted a *mercury manometer for* Hales tube and in 1847 Ludwig[7] devised a direct recording manometer Subsequent studies are numerous[1, 3, 5, 10, 11, 13, 17, 2, 5, 36]

In 1896 Riva Rocci[41] described an indirect method for blood pressure estimation based on studies of several researchers[0, 30, 8, 34, 39, 47] This method included the use of a pneumatic cuff for compressing the arm and palpation of the radial artery The first palpable pulse wave appearing during decompression marked systolic pressure

Bing[4] Pal[34] Vaquez[47] and others devised a double cuff as well as visual oscillatory indicators and a double cuff was also used by Groedel and Miller[0]

These studies and the investigations of Marey[0] and Recklinghausen[40] paved the way for the first practical oscillatory sphygmomanometer by Pachon[3] This apparatus is composed of an aneroid manometer and of a constantly balanced capsule indicating the amplitude of the arterial oscillations As the cuff pressure is lowered small oscillations appear (supramaximal oscillations) followed by an abrupt increase of amplitude (systolic pressure)

The oscillations increase progressively to a maximum, then decrease, first gradually, then abruptly and are followed by other small oscillations (in framinimal oscillations)

The diastolic pressure was first thought to coincide with the maximal oscillation as the arterial wall should oscillate freely between two equivalent pressures[30] Later on, as a result of the investigations of Macwilliam and Melvin,[29] and of others[15 18 33] the diastolic level was placed where a marked change in amplitude occurs Recording oscillometers were devised by Plesch[35] (tonoscillograph) and Boulitte[6] (arterial oscillograph)

In 1905 Korotkow[4 25] suggested that blood pressure may be estimated by auscultation of the brachial artery

Subjective differences in the evaluation of the blood pressure have been appreciated since 1881 As a result several graphic methods based on the sphygmographic principle have been devised[6 7 12 14 16 23 31 35 4 43] More recently, Gomez and Langevin[19] utilized a piezoelectric quartz crystal and an electronic amplifier to register the pulse However the advantages of the piezoelectric crystal were lost because the pulse was transmitted to the crystal through a contact button

Von Recklinghausen[40] observed that graphic tracings of the pulse during deflation of the cuff from above systolic pressure to below diastolic show minute changes in the shape of the pulse waves which indicate the lateral and the end pressures in the systolic and diastolic phases Bazett and Laplace[3] confirmed these observations

Following these studies Rappaport and Luisada[38] devised an apparatus which registers all physiologic phenomena to be evaluated for estimation of blood pressure by the palpatory oscillatory and auscultatory methods

INDIRECT SPHYGMOMANOMETRY

Technic

The apparatus of Rappaport and Luisada[38] consists of a double chamber pneumatic cuff covered by nondistensible material The upper is the *pressure chamber* and is of the usual dimensions (13×25 cm), the lower is the *registration chamber* and is 3×25 cm (Fig 74) The pressure chamber is connected to a manometer a needle valve and an inflation pump This part of the system may communicate with or be cut off from the rest by the setting of valve "A

The essential part of the system consists of two differential crystal microphones Such microphones are normally incapable of withstanding any appreciable pressure without damage to the crystal However the pressure is allowed to communicate with the front and rear of the diaphragms when valve B" is in the open position As these pressures are identical the

crystals are not deformed The arterial pulse applied to the registration chamber of the cuff distends the diaphragms and bends the crystals in proportion to the change of pressure

The double chamber cuff is applied to the subject as with the usual type of sphygmomanometer With valves A and B open the system is inflated by means of the pump all pneumatic channels are in communication under the pressure indicated by the manometer When a pressure of about 20 mm Hg is attained valves A and B are closed

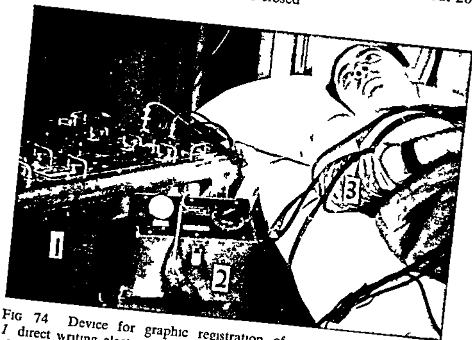


Fig 74 Device for graphic registration of arterial blood pressure *1* direct writing electrocardiograph *2* box containing the crystal microphones *3* double cuff

The pressure is now increased in the pressure chamber of the cuff to above the anticipated systolic pressure then opening of the needle valve leads to its gradual deflation At this point the registration chamber detects both the Korotkow sounds and the pulses and transmits them to the microphones The microphone for sounds is a stethoscopic microphone (p 30) that for pulses a linear microphone (p 28)

Analysis of the Tracing

When a pneumatic blood pressure cuff is inflated to a pressure above systolic and allowed to deflate gradually the pulsation of the artery is modified (Fig 75) As long as the cuff pressure is above the systolic level the artery is collapsed and no pulsation is detectable below the cuff When the cuff

pressure is slightly less than systolic a small pulsation appears, this represents the uppermost portion of the pulse the pressure level of which exceeds cuff pressure, allowing the collapsed artery to open slightly during a short interval When the cuff pressure is further lowered, a larger portion of the pulse passes through the artery because the collapse of the latter occurs only during a shorter period of time When the cuff pressure approaches the diastolic level *the pulse is completely transmitted and there is no arterial collapse*

Thus, the systolic pressure of an artery may be estimated by a sphygmo graphic device capable of detecting the pulsation just below the pressure cuff The systolic level is the point at which the first pulsation is detected when the cuff pressure is gradually lowered from above the systolic level

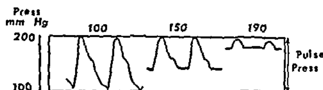


FIG 75 Theoretical configuration of the pulse curve in two succeeding cycles at different values of *cuff pressure in a patient with a blood pressure of* 200/100

Two different methods have been advocated for the determination of dia stolic pressure by the Kor otkow method The first was advocated by the Anglo American Commit tee of 1939,[43] the second by the Committee of the American Heart Associa tion of 1951 [44] The first maintained that the level of diastolic pressure should be placed where the sounds become dull and muffled a view shared by the author The second, that diastolic pressure should be placed where the sounds disappear

As the cuff pressure gradually drops from above the systolic level the re corded pulsations become larger and larger but a rather flattened base line is present between adjacent cycles until the diastolic level is reached The cuff pressure at which the first pulse appears with a well defined and peaked contour is the diastolic level

The Korotkow sounds occur simultaneously with the sharp primary oscilla tion of the pulse or during initial ejection of blood into the artery During the rapid ejection the blood velocity is greatly increased by passing through the partially collapsed portion of the artery immediately below the pressure cuff This sets the artery into vibration resulting in the creation of a sound As the cuff pressure is gradually lowered the arterial pulsations and the Korotkow sounds increase in intensity (Fig 76) The Korotkow sounds suddenly dimin *ish in intensity and thereafter are less and less intense This sound effect can* be explained as follows when the cuff pressure is below the diastolic level there is no longer constriction of the artery also the rapid distention of the vessel by the primary oscillation takes place in a less and less confined area as the cuff pressure is lowered

Another sound, commonly registered in the blood pressure tracing occurs simultaneously with the notch preceding the dicrotic wave The mechanism of production of this sound is identical to that of the Korotkow sounds Its intensity is much less than that of the former because the energy generated by the sudden drop in pressure is obviously of lesser magnitude

Femoral systolic pressure cannot be measured accurately with the ordinary 13 cm cuff A wider cuff (15 5 cm) permits more accurate measurements except in patients with aortic regurgitation On the other hand femoral diastolic pressure seems to be inaccurate even with the larger cuff [26]

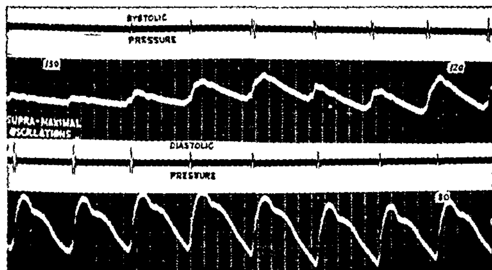


FIG 76 Graphic tracing of arterial pressure by indirect sphygmomanometry *above* arterial sounds *below* arterial pulses

DIRECT SPHYGMOMANOMETRY

Technic

Direct sphygmomanometry is based on puncture of an artery and direct connection of its lumen with a graphically recording manometer [6, 10, 11, 17, 19, 36] The arteries usually employed are the radial and the femoral Puncture of these is easy because they are superficial and have a solid bony support The brachial artery can be used also but its puncture requires more care because the vessel moves easily under the skin

The needle used for insertion in an artery is fitted to a 2 cc syringe and, through a 3 way stopcock to the manometer The syringe is filled with heparin Following superficial anesthesia with 2 per cent novocaine, the artery *is penetrated As soon as blood spurts into the syringe and mixes with the* heparin pressure is exerted so that the needle fills with the anticoagulant

Then the stopcock is turned and the arterial pulsations are transmitted to the manometer

Any type of manometer can be used In the past a Hamilton manometer was usually employed At present the best type is an electromanometer* which is connected with the electrocardiograph before starting the procedure Calibration is done and recorded on the film so that the amplitude of the deflections and the levels of systolic and diastolic blood pressures can be measured A switch on the apparatus permits taking either a tracing of mean pressure or a tracing of the pulsating changes of blood pressure The record is taken at a slow speed if one is interested in the slow variations of pressure at a film speed of from 50 to 75 per second if one wishes to study the shape of the pulse waves (Fig 77) Either the photographic or the direct writing methods can be used

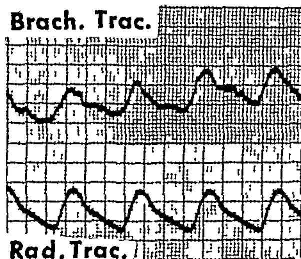


FIG 77 Brachial pulse (indirect method) radial pulse obtained by introducing a needle into the radial artery (direct method)

After completion of the record the stopcock is turned and the needle is rapidly extracted while pressure is exerted on the artery above the site of puncture It may be necessary to keep the artery compressed for one or two minutes After this time no hemorrhage is to be feared A small piece of sterile gauze and a strip of adhesive complete the procedure

CONCLUSIONS

Indirect graphic sphygmomanometry is used whenever doubts arise about blood pressure measurements In particular cases with an auscultatory gap or with an indecisive diastolic pressure may require graphic tracings Patients with coarctation of the aorta, arteritis or embolism of the limbs, thrombosis of the abdominal aorta, or functional disturbances of the peripheral vessels may require these tracings

It is generally agreed that the direct method is the most accurate for measuring blood pressure However possible errors due to nonabsolute transmission of pressure from artery to instrument spastic contraction of the artery in

* A currently used type of electromanometer is supplied by the Sanborn Co of Cambridge Mass (Appendix D)

which the cannula has been introduced psychic factors and the difficult determination of an exact zero level may exist Although this method is invaluable in the physiologic laboratory the procedure of inserting a cannula into the artery of a patient is a decided limitation from a clinical standpoint

VENOUS PRESSURE

Direct measurement of venous pressure can be done with the same technic as that used for direct measurement of arterial pressure A vein of the forearm is selected and care should be taken that the vein is at a definite level

According to Burch [8] a *phlebostatic axis* should be determined first This is defined as the line of junction between a transverse plane of the body passing through the points of junction of the lateral margins of the sternum and the fourth intercostal space and a frontal plane of the body passing through the midpoint of a line extending from the outermost point of the anterior surface of the sternum to the outermost point of the posterior surface of the chest Then a phlebostatic level should be found Any plane which passes through the phlebostatic axis and is also parallel to the horizon is a phlebostatic level This corresponds to the heart level and is to be considered as having zero pressure Any figure above or below it represents an actual variation of venous pressure

Before proceeding to the recording the electromanometer is set at a high sensitivity level and calibration is made The record is taken at low speed if one wishes to study respiratory or slow changes of venous pressure at speeds of 50 to 75 mm per second if one wishes to study rapid variations of pressure (Fig 78)

Conclusions

Graphic registration of venous pressure is seldom necessary for diagnostic purposes On the other hand the method has useful applications for studies of cardiovascular dynamics

PULMONARY ARTERIAL PRESSURE

Pulmonary arterial pressure in man is more commonly recorded by means of right heart catheterization (p 163) Following direct recording in a clinical case by the author another method has been advocated by Vacarezza *et al* [46] These authors introduced a needle in the second left interspace 1 cm outwards of the sternal margin and under fluoroscopic control punctured the trunk of the pulmonary artery This vessel was encountered at 5–6 cm of depth (4 in adolescents 8 in cases with emphysema) There were no after

effects If this technic is employed the procedure is that already described for direct arterial pressure records

Conclusions

Direct puncture of the pulmonary artery should *not* be *performed unless* further studies prove that it is completely devoid of dangers Should this be proven then the method might lend itself to practical applications in the field of congenital heart disease, cor pulmonale, and various other heart diseases

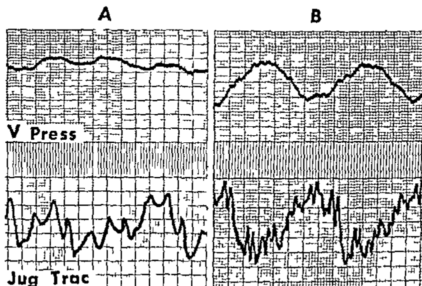


FIG 78 *Above* tracing of venous pressure in a vein of the fore arm (direct method) *Below* jugular tracing *A* higher film speed normal respiration *B* lower film speed, forced respiration

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CHAPTER 19

Tracings of Intracardiac and Intravascular Pressure

(CARDIOMANOMETRY)

DIRECT MEASUREMENT

History

Direct measurement of pressure in man through puncture of the heart has been done in either ventricle[1, 14] and in the pulmonary artery [4, 29] This is done only in special clinical cases and the method should not be considered for routine use because of its dangers

Forssman[9, 10] was the first to pass a catheter into the right heart of man doing it first on himself with the help of a colleague Heuser[15] Klein[17] and Conte and Costa[2] used the method for roentgenologic studies or for determination of cardiac output Cournand and coworkers[3, 6] later used this method on a large scale and perfected the technic Dexter[7, 8] Sossman[28] Lenegre[19, 0] McMichael [5] and many others have published important contributions

RIGHT HEART CATHETERIZATION

Technic[4, 8, 0]

The procedure is carried out by a special team The patient lies supine on a fluoroscopic table covered by a pad Mild sedation is used in the adult general anesthesia in the child and infant The usual protections against excessive

radiation both to the patient and the observer, are used However, the operator wears no lead gloves and should avoid exposure of the hands to x ray

A No 6F or 7F catheter 100 cm long made of woven fabric with a plastic covering,* is used A small curvature near the tip permits direction of its progress through rotation of the external end The latter is fitted with an adaptor similar to the hub of a hypodermic needle

The catheter can be sterilized by autoclaving or by boiling for ten minutes in distilled water After sterilization it is wrapped in a sterile towel and should be tested for surface imperfections or leaks before introduction After use it should be rinsed with tap water, filled with "Haemo sol solution and allowed to soak in the same solution for one hour Then it should be rinsed for one hour with tap water through a pressure connection This procedure serves to prevent deposition of particles of blood †

A sterile tray carries the instruments necessary for skin incision, isolation of the vein and closure ‡

An electromanometer¶ is prepared and calibrated The entire tubing is filled with saline and a small amount of heparin is injected so that it fills the catheter The bottle of the electromanometer can be filled with saline plus heparin, or with citrate solution

Any vein of either the right or the left arm can be used The ideal site is below the confluence of the median basilic vein with the other branches running medially to it Vision of the field by the patient is prevented by a small screen The skin is sterilized, then it is infiltrated with 2 per cent novocain and a sterile tourniquet is applied above the elbow A transverse incision about 1 cm long is made (a longitudinal cut may obviate the need for suture) The vein is isolated by blunt dissection and a double loop of silk is passed under it The silk is divided and one piece pulled to the distal end of the exposed part and tied with a single knot to prevent retrograde bleeding The catheter and its attachments are then prepared

The vein is elevated and a small fish mouth incision is made with a bayonet type scalpel The catheter tip is then inserted in the vein and passed several centimeters up the lumen Further progress of the catheter is carried under fluoroscopic control If obstruction is met on attempting to enter the chest rotation of the catheter or slight movements of the arm and shoulder usually

* Supplied by U S Catheter and Instrument Co Glens Falls N Y

† According to L. Dexter (*Proc Soc Exp Biol & Med* 79 444 1952) sterilization of the catheter may be responsible for pulmonary infarction if done by means of antiseptics

‡ They are the following two sheets and two large towels two towel clamps two small curved and two small straight mosquito hemostats a plain forceps a scalpel with an N 11 blade a small scissors a medicine glass for the novocaine solution two No 26 needles for local anesthesia two 5 cc syringes a curved cutting edge needle with black silk for closure of the incision a small roll of black silk N 0000 for ties several 3 way stopcocks and extra adaptors a large beaker to hold saline solution a 50 cc Luer lock adaptor syringe for testing the catheters for leaks a tourniquet sterile gowns gloves sheets and infusion

¶ Supplied by the Sanborn Co of Cambridge Mass (Appendix D)

allows it to proceed The catheter is directed first into the superior cava then into the right atrium At times, the tip enters the inferior cava then, the catheter should be withdrawn and reinserted Once the catheter is in the right ventricle further passage into the pulmonary artery is usually possible by means of gentle rotations and movements of withdrawing and insertion The tip then is pushed into one of the main stems and further on into the fine pulmonary ramifications

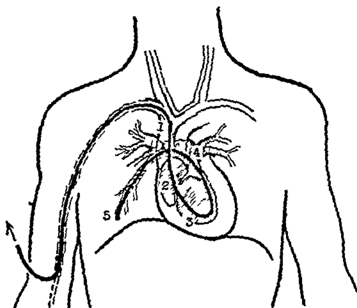


FIG 79A Scheme of right heart catheterization Course and location of the catheter *1* superior cava *2* right atrium *3*, right ventricle *4* pulmonary artery *5* pulmonary arteriole (for capillary pressure)

As venous spasm may prevent introduction of the catheter it is advisable to push this as far as possible and to proceed to the various pressure measurements and oxygen determinations during withdrawal The following records can be taken (a) pulmonary capillary pressure [7, 8] (b) pulmonary artery pressure (c) right ventricular pressure (d) right atrial pressure (e) superior caval pressure (Fig 79A) The records should be taken at a film speed of 50 to 75 mm per second in order to obtain sufficient spreading of the waves however if one is only interested in pressure readings any lower speed is adequate

On completion of the procedure the catheter is withdrawn and the two silk ties are removed The skin is closed with two fine silk sutures and bleeding

is controlled by a pressure bandage The sutures are removed several days later

Oximetry

Oximetry at present is done by evaluating chemically or photoelectrically the oxygen content and saturation of samples of blood drawn through the catheter It is likely that within a few years oximetry will be done graphically and automatically from the tip of the catheter and without aspiration of blood For this reason, the necessary formulas are given here

Evaluation of oxygen saturation of blood samples collected during heart catheterization (oximetry) supplies essential data for calculation of both the systemic and the pulmonary blood flow The existence of a shunt and the amount of blood flowing through it, can also be determined

The estimated systemic blood flow (*Qs*) is equal to oxygen consumption in the tissues (i e oxygen intake in the lungs) divided by the difference between oxygen concentration in the aorta or peripheral artery* (*Cao*) and in the right atrium (*Cra*)

The estimated pulmonary flow (*Qpa*) is equal to oxygen consumption divided by the difference between oxygen concentration of the blood of the left atrium (*Cla*)† and that of the blood in pulmonary artery (*Cpa*)

$$Qpa = \frac{O_2}{Cla - Cpa}$$

When the oxyhemoglobin concentration of the arterial blood is decreased far below the normal figure of 96 per cent, either a right to left shunt or pulmonary pathology will be suspected

Various calculations can be made upon data supplied by cardiomanometry[18] The most important are (1) determination of mitral area, (2) determination of mitral regurgitant area (3) determination of regurgitant flow

These determinations involve complex mathematical formulas and their accuracy has not been established as yet If confirmed they have a special importance in mitral valve lesions

Complications

Early accidents[19] are represented by arrhythmias chill and acute pulmonary edema The last has been observed following infusion of saline or rapid digitalization It should be treated by injection of a mercurial diuretic aspiration of 500 cc of blood, and administration of alcohol vapor by inhalation[27]

* The blood sample is usually drawn from the brachial radial or femoral artery

† This is of course identical with that of a peripheral artery if there are no shunts between the ventricles or the large arteries

Disturbances of the heart rate and rhythm are common Premature contractions are frequent when the tip of the catheter is near the tricuspid valve or against the ventricular septum Bouts of ventricular tachycardia have been observed when the tip of the catheter passed through a ventricular septal defect Atrial tachycardia has been reported in cases of atrial septal defect Right bundle branch block and atrial fibrillation have been observed Normal hearts seem to present these complications as often as diseased hearts In the cases with nonfatal arrhythmias the disturbance usually disappears within twelve hours [11] However, ventricular fibrillation should be anticipated so that immediate treatment is undertaken

Sudden death is an extremely rare event reported in about 0 1 per cent

Venous thrombosis for 5 to 7 cm from the site of section of the vein is customary However, the vein may be recanalized later If the site selected was at least 5 cm from the last confluence of the veins of the basilic system the flow of the main brachial vein is not interfered with

Pressure Tracings and Their Interpretation

SUPERIOR CAVAL TRACING Published tracings are frequently distorted by artifacts They usually present a typical venous appearance (p 98) The customary three waves *a c* and *v* are well defined Small vibrations may occur during early systole and at the time of the second heart sound Pressures vary between 0 and + 10 mm Hg (Fig 79B)

RIGHT ATRIAL TRACING This tracing resembles that of the superior cava it shows a tall (a) wave in presystole (atrial contraction raises the pressure), a systolic collapse (ventricular contraction pulls downward the tricuspid valve causing suction) and a gradual rise culminating with the (v) wave at the time of tricuspid valve opening The (c) wave is usually absent, when present it takes place during the tension period and is caused by a slight raising of the tricuspid valve Small vibrations may be present in early systole and at the time of closure of the semilunar valves Pressures vary between 0 and 8 mm Hg with a mean pressure of 1–2 mm Hg (Fig 79B)

RIGHT VENTRICULAR TRACING This is a typical curve of intraventricular pressure (Fig 79B) A small positive wave is present in presystole due to completion of filling by the atrial contraction A rapid rise of the curve takes place during the tension period and terminates with a small notch which marks the closure of the tricuspid valve After further rise a second notch marks the opening of the pulmonic valve During ejection a steady line is typical (plateau like curve) but a rounded contour may be observed A sudden drop indicates the end of ejection This is followed by two vibrations the first marks the closure of the pulmonic valve the second, the opening of the tricuspid valve Rapid filling is usually not accompanied by rise in pressure proving that the right ventricle dilates gradually with the incoming blood Pressures vary between 35 mm Hg (systolic) and zero (diastolic)

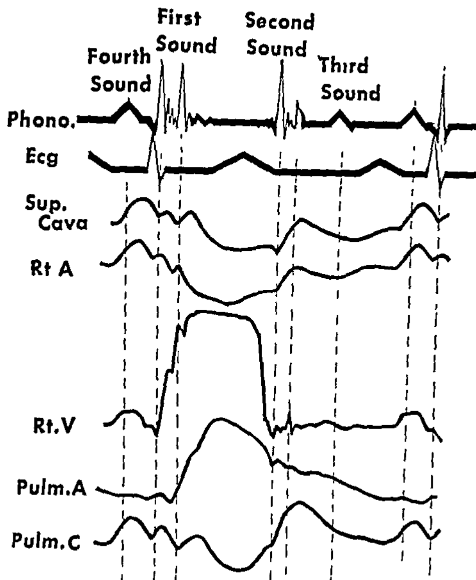


FIG 79B Catheterization of the heart Comparison of the pressure pulses with the electrocardiogram and phonocardiogram *Rt A* right atrium *Rt V* right ventricle *Pulm A* pulmonary artery, *Pulm C* pulmonary capillary" pressure

PULMONARY ARTERY TRACING This is a typical arterial tracing (p 137) The anacrotic notch is usually well visible The peak is rounded (except if artifacts mar the record with superimposed vibrations) The incisura is deep The dicrotic wave is well visible and followed by a few aftervibrations Pressures vary between 30 mm Hg (systolic) and 10 mm Hg (diastolic) (Fig 79B)

PULMONARY CAPILLARY TRACING This is essentially a venous tracing[13] the waves being due to changes of pressure transmitted from the pulmonary veins through the capillaries of the lungs The tracing shows a positive atrial wave an early systolic dip followed by a systolic collapse and an early diastolic rise a mid diastolic collapse Differences between this tracing and that of the right atrium are a higher mean pressure a delay of the waves of about 0 08 second a deeper diastolic collapse Mean pressure is about 3 mm Hg (Fig 79B)

Tracings of the left atrium have been recorded in cases of atrial septal defect by passing the catheter through the defect

CATHETERIZATION OF THE LEFT HEART OR AORTA

Catheterization of the human aorta has been used for the study of the aortic pressure pulses[7] The procedure seems to be no more hazardous than catheterization of the right heart The contour of the pulse closely resembles that of the aortic pulse in the dog

Catheterization of the left heart has been attempted in man first in cases with aortic regurgitation then in normal subjects[18] The technic is based on isolation of the left brachial artery introduction of the catheter into the artery penetration of the catheter into the aortic arch then through the aortic valve into the left ventricle The procedure is contraindicated on account of the dangers which it entails These consist of rupture of the aorta, occlusion of a coronary artery rupture of an aortic leaflet attacks of ventricular tachycardia or flutter The tracing of pressure of the left ventricle is similar to that of the right ventricle in regard to the various details The systolic pressure is similar to that of the aorta the diastolic pressure is zero in normal subjects

CONCLUSIONS

Together with determination of oxygen content measurement of pressure within the chambers of the right heart and the pulmonary vessels is of great importance in the diagnosis of congenital heart diseases Low speed tracings are used in order to obtain an objective determination of pressure High speed tracings are useful for the study of the various details of the curves and for solving problems of cardiac physiology in the normal and diseased heart Important observations have been made in congestive failure chronic cor pulmonale tricuspid valve defects and constrictive pericarditis The tracing may have a diagnostic value in the last two conditions

Left heart catheterization is dangerous and should not be attempted

Catheterization of the aorta may present interest in coarctation of the aorta

INDIRECT MEASUREMENTS OF PRESSURE

Several attempts have been made in order to evaluate the pressure of the lesser circulation by means of electrokymography This method (p 178) has been applied to the study of the pressure in the main trunk (Kay and co workers[18]) or in the small branches of the pulmonary artery (Marchal[26] Gillick and co workers[1]) It is the impression of the author that the method is not reliable in either case This impression was confirmed for the pulmonary arteriolar pressure by personal studies -

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SECTION G

Radiography of the Motions and Volume Changes of the Heart and Large Vessels

CHAPTER 20

Tracings of Cardiovascular Pulsations on the Roentgenogram

(ROENTGENKYMOGRAPHY)

HISTORY

The first roentgenkymograms were recorded by Goett and Rosenthal[4] in 1912 Hitzenberger and Reich[6] described a somewhat different method in 1923 Chamberlain and Dock,[2] a few years later, attempted to record the motion of the cardiovascular silhouette by means of cinematography In 1931 Stumpf[11] constructed a new type of screen Zdansky and Ellinger,[1] Cignolini[3] Heckmann Morelli and Trouillier,[8] and Lenzi[7] brought further contributions In 1937 Heckmann[5] and Bordet and Fischgold[1] published the first monographs on the subject Most textbooks of roentgenology, like those of Roesler[9] and Schwedel[11] give detailed descriptions of roentgenkymographic technics and of the various tracings

TECHNIC

The roentgen ray permits direct visualization of cardiovascular silhouettes and yields accurate records Tracings of the motion of the cardiac silhouette are called *roentgenkymograms*

If the lead diaphragm of the fluoroscope is narrowed to a thin slit, single points of the cardiac border can be seen to pulsate inward and outward with cardiac systole and diastole If an x ray film is substituted for the fluoroscopic screen and either the film or the grid is made to move at constant speed the pulsation of a point of the border is recorded as a section of a curve (Fig 80)

Goett and Rosenthal[4] used a single slit while Hitzenberger and Reich [6] and Zdansky and Ellinger [12] used two slits Stumpf[11] constructed a screen containing several horizontal slits at a distance of 12 mm from each other so that

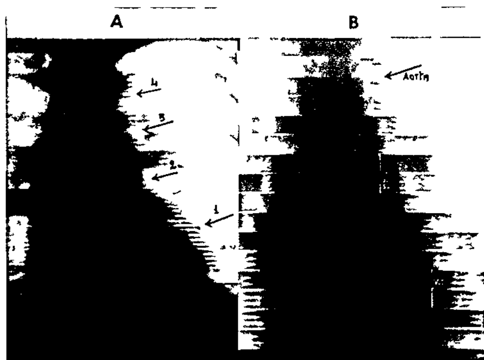


FIG 80 Roentgenkymograms (A) System with moving slits (B) System with moving film

kymograms of the entire cardiovascular silhouette were obtained Cignolini[3] invented another multiple slit device where several slits could be adjusted over points of greatest interest on the cardiac silhouette Morelli and Trouillier[8] developed a type with radial slits and a rotatory motion of the film (Fig 81)

ANALYSIS OF THE WAVES

Atrial ventricular and vascular waves can be observed To interpret them one should turn the film 90° counterclockwise and put it in front of a mirror

for the study of the left cardiac border, 90° clockwise, for the study of the right cardiac border This difference between the two borders represents the first obstacle in the interpretation Each trough is the result of an inward movement (systole contraction depression) each crest is caused by an outward movement (diastole expansion, pulsation) (Fig 82)

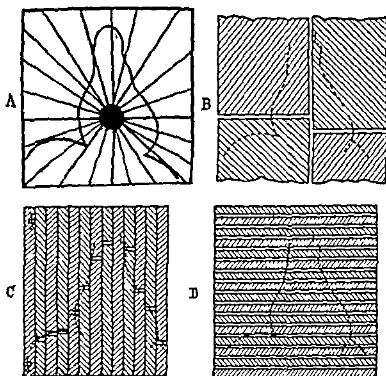


Fig 81 Scheme of various devices for roentgenkymography *A* radial slits (Morelli and Troullier) *B* two adjustable slits (Zdansky and Ellinger) *C* multiple adjustable slits (Cignolini) *D* fixed horizontal slits (Stumpf)

A complete description of the waves will be given in the next chapter because the waves revealed by the electrokymograph are the same as those of the roentgenkymograph The latter, however are smaller less sharp and less well defined

Anatomic Considerations

The limitations of roentgenkymography are of two kinds those due to anatomic and physiologic conditions and those inherent in the technic The anatomic and physiologic limitations are (1) A linear movement can be

registered graphically in its true excursion only if observed at a right angle otherwise the excursion appears smaller than it really is (2) Only the pulsation of the visible contours may be registered so that no conclusion as to the total change in volume of a chamber may be drawn (3) Pulsations as well as changes in volume of the cardiac chambers and large vessels are transmitted to adjacent structures this is particularly true of the transmission of ventricular pulsations to the atria The pulsations observed at the surface of these struc

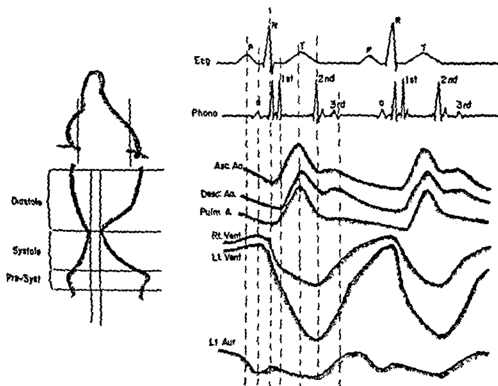


Fig 82 Reconstruction of the pulsations of the various points of the cardiovascular silhouette Both right and left sided tracings are reconstructed spread and superimposed so that they can be read from left to right

tures often represent a summation of their own weak pulsation with transmitted pulsations (4) The heart performs several types of movements such as rotation lateral shift and elevation of the apex Also the effect of respiration on heart motion must be taken into account These movements distort and modify the waves caused by changes in volume of the chambers (Fig 83) These considerations make it clear that the amplitude and even the direction of individual pulsations may be subject to complicated distortions and that

any study of the tracing must take into consideration all sources of interference

Technical Considerations

The technical limitations in the use of roentgenkymography are (1) Difficulty of applying the apparatus in a position perpendicular to the contour to be plotted because of the rigidity of the slits (2) Short duration of the records obtained (only three or four cycles in Stumpf's multiple slit kymograph) (3) Insufficient speed of the tracings (4) Poor distinction and small amplitude of the waves (5) Inability to record simultaneously other tracings like an electrocardiogram a phonocardiogram or a sphygmogram Attempts to overcome these weaknesses by faster recording over a longer period of time[3,1] or densometric transcription[11] have not resulted in a great improvement

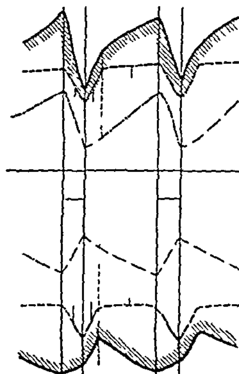


Fig 83 Scheme of the motion of the left (above) and the right (below) heart borders in the roentgenkymogram according to Heckmann The shaded line represents the actual tracing resulting from volume changes (dot-dash line) and positional changes (broken line) (From H Roesler courtesy of Charles C Thomas Publisher)

CONCLUSIONS

Roentgenkymography gives permanent and simultaneous records of the motions of several points of the cardiovascular silhouette It permits analysis and detailed study of the pulsations as to amplitude and time It may still be of interest in aneurysms mediastinal tumors coarctation of the aorta and pericarditis In general the importance of roentgenkymography has been decreased by electrokymography which is handier and more accurate (p 178)

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CHAPTER 21

Tracings of Cardiovascular Pulsations on the Fluoroscope

(ELECTROKYMOGRAPHY)

HISTORY

The first attempt to record fluoroscopic phenomena by phototubes made by Hjelmare[1] in 1932 was unsuccessful Heckmann[8] tried in 1937 a similar method which he called actinography In 1945 Henny and Boone[10] developed the first practical apparatus and called it electrokymograph They applied it to the study of border motions of the right atrium the aortic and pulmonary knobs and the left ventricle A year later Marchal[-3] built independently a similar device for the study of density changes of the lungs and called it kinedensigraph In the same year Lian and Minot[13] used another similar device for the study of the right and left heart borders Henny Boone and Chamberlain[11] modified the characteristics of their apparatus† in 1947 and studied density changes of the heart and vessels In the same year the author, working with Fleischner and Rappaport[19 0] described an instrument which operated on the same principle as that of Henny and Boone but combined several modifications ‡ With this a systematic study of the border

* Alternate terms suggested *fluorocardiography* [0] *kinedensigraphy* [3]

† This is supplied by the Cambridge Instrument Co of New York

‡ This was built by the Sanborn Co of Cambridge Mass

motions and density changes of the heart great vessels and lungs was made in several positions of the subject [10, 1] Subsequent studies of electrokymography dealt with the time relationships of the contractions of the right and left heart (Chamberlain *et al* [4] Luisada and Fleischner [16]) the isometric relaxation phase (Randak *et al*,[3, 6] Luisada *et al* [2] Mednick *et al* [1]) the relationship between electrokymograms and curves of intracardiac pressures (Luisada and Fleischner [17] Salans *et al* [9]) and the technic of recording during normal respiration (Luisada and Fleischner[18]) Among the numerous subsequent studies that of Engstroem and co workers described another valuable apparatus

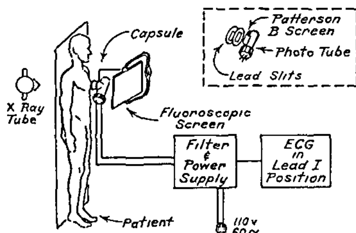


FIG 84 Scheme of the arrangement for electrokymography

TECHNIC

The set up for electrokymography (Fig 84) consists of (1) fluoroscope (2) pickup fastened to the fluoroscopic screen (photo multiplier tube slitted diaphragm and small fluorescent strip) (3) electrical arrangement to transform AC into DC and device for rapid changes of amplification (4) electric filter suppressing the flicker of the x ray tube (5) galvanometer transcribing the tracing on a moving film, (6) microphone to be connected with a sound recorder, (7) device for electrical tracing of the carotid pulse

Any upright fluoroscopic stand can be used for the seated patient for children or sick patients a horizontal table is used A commercial tilt table can be employed for both purposes

The main part of the apparatus is an electron multiplier phototube * This

* The tube is produced by the Radio Corp of America Camden N J At first the author used a 931 A subsequent experience showed that a 1P21 phototube gives even better results

consists of a light sensitive *photocathode* nine secondary electrodes called *dynodes* and a collector *anode* The phototube has a maximal response to a wave length of 4200 Å which is in the blue region of the spectrum, while it gives an approximate 70 per cent response to green light

When the photocathode is exposed to light a proportionate flow of electrons is released and attracted to a dynode where it displaces additional electrons The secondary electrons are directed to a second dynode (which is at a potential more positive than the first) where they displace more electrons Since this process of multiplication is cumulative in the nine stages of amplification the maximum over all amplification reaches about one million times (two million times for the 1P21)

A strip of *Patterson B fluorescent screen* is cemented to the glass cover of *the phototube in front of the cathode When the x rays strike this screen the* light generated is picked up by the cathode transformed into equivalent electrons, amplified by multiplication, and fed into the electrocardiographic channel Thus the electrical output varies in proportion to the amount of x rays which reach the fluorescent screen cemented to the phototube

The phototube is protected by a lead screen with a slit 5×25 mm If the slit is placed across the border of the cardiovascular silhouette the amount of x rays received by the small fluorescent screen varies with the motion of the border (*border tracing*) If the slit is against a shadow, the amount of x rays *varies with changes in transparency of the shadow due to changes in thickness* of the structure (*densogram*)

Three forms of interference must be eliminated (a) electrostatic radiation from the high voltage x ray equipment (b) flicker due to cyclic discontinuity of the x ray emanations and (c) fluctuations of the power line

Elimination of interference caused by the x ray equipment can be obtained by electrostatic shielding of the electrokymographic circuit

The anode potential supplied to the x ray tube may be obtained by full wave or half wave rectification If the frequency of the power line is 60 cycles per second a full wave rectifier supplies 120 pulses per second while a half wave rectifier supplies only 60 pulses per second Thus the fluorescent screen flickers either 120 or 60 times per second depending upon whether full or half wave rectification is used To make possible the registration of small border movements or slight density variations which are imperceptible to the eye an electric filter attenuating the flicker at least 100 000 times is used

The polarity of the apparatus is so arranged that an increase of light causes a downward movement of the tracing Therefore a drop of the curve indicates either an *inward motion* of the cardiac border (border tracing) or a *decrease in the thickness* of the structure (densogram) The former may be due to either contraction or displacement

Any wave occurring before the first large vibration of the first sound is

presystolic, any wave occurring after the last vibration of the second sound is diastolic, any wave taking place between the beginning of the first sound and the end of the second is systolic

If the galvanometer has a deflection speed of 0 01 second the effective speed due to the tailing off of the filter when this is tuned to 120 cycles per second, is approximately 0 02 second

If an electrocardiogram is to be taken simultaneously with the electrokymo gram special attenuators must be interposed between the subject and the electrocardiograph Otherwise, the unshielded patient picks up electrostatic radiations from the high tension components of the x ray and there will be artifacts in the electrocardiogram

The graduated potentials applied to the nine dynodes of the phototube are obtained from the AC power line The fluctuations of the commercial power lines must be regulated or smoothed out before reaching the phototube

Timing of the waves can be obtained by the simultaneous recording of the eky with a carotid tracing a phonocardiogram or both The last procedure is the best whenever a three channel electrocardiograph is available If one records the carotid tracing this should be taken by means of a linear micro phone avoiding therefore mechanical delay In the author s laboratory the carotid tracing is recorded by means of a cuff wrapped around the neck (p 154), the phonocardiogram is recorded by a stethoscopic microphone fastened to the fluoroscopic screen and connected by a short tube to a trans parent bakelite funnel held against the chest by a rubber strap

Calibration

Calibration of electrokymograms is very important Comparison of tracings recorded over different structures or over the same structure at different times may be made only through use of calibrating devices These indicate whether or not the change in amplitude of a wave corresponds to different amplitude of a border motion (or to different density) Various devices for calibrating have been built One of them calibrates density changes [5] another, amplitude of border motions [9] [3] However any tracing of border motions contains also a component of density change acting on that part of the slit which is within the shadow This component cannot be excluded and cannot be separately evaluated Therefore it is the opinion of the author that, at present one single device modifying the intensity of x ray received by the fluoroscope and calibrating the sensitivity of the instrument is the only one having practical utility

Film Speed

A good analysis of the waves can be made only if the film moves at speeds of between 50 and 100 mm per second If on the other hand one is interested

in the magnitude of the waves, film speeds of between 2 5 and 10 mm per second should be used (Fig 101)

Working Procedure

The pickup device is attached to the fluoroscopic screen by means of a brace It is centered so that it is fully exposed to the x ray beam with the diaphragm narrowed down to a small field It can be fastened to a special screen according to the description of Grossman and Tiger [7]

The procedure is performed by a two man team The recording apparatus the electrical unit and the pickup device are assembled and the wire con nection attached The patient is seated on a rotating stool in front of the fluoroscope and an elastic strap is slung around his chest to hold the funnel of the microphone the pneumatic cuff is wrapped around his neck Carotid and sound tracings are checked and the patient is instructed on how to hold his breath *The room is darkened for fluoroscopy screened lights illuminating* the panels of the instruments

With the patient in the posteroanterior position, the pickup is placed under fluoroscopic control at the cardiac apex with the slit perpendicular to, and crossing that part of the silhouette to be studied With the slit in place and the fluoroscope in operation the operator regulates the amplitude of the de flections asks the patient to hold his breath and starts the camera keeping under observation the light beam After obtaining a tracing of several heart cycles the operation is interrupted the pickup is moved to the next place and the procedure is repeated The position of the patient and that of the pickup as well as the degree of amplification used are noted for every tracing

Ordinary technic for chest fluoroscopy is used with 3–5 ma and 65–70 Kv Initial fluoroscopy with the open shutters takes only a few seconds then the shutters are narrowed to a small opening As actual total recording requires from three to five minutes exposure to x ray is moderate

In general the tracing should be recorded during voluntary apnea in an intermediate phase because it has been shown that the pulsations of the lung (and *to a lesser extent those of the hilar shadows and the pulmonary artery*) are greater in inspiration than in expiration After a few words of instruction usually the patients learn to hold their breath in an intermediate phase It is difficult however to obtain reliable tracings in children patients in failure and patients with chronic lung diseases who are unable to control their respi ration because of age or dyspnea This difficulty can be overcome by altering the electrical time constant of the apparatus A high pass filter made of several condensers is interposed between the electrokymograph and the galvanometer, this filter modifies the time constant so that the tracing does not wander off the paper A slight error proportional to the amount of re duction and consisting of a slight change of phase and configuration of the

waves is the result Therefore such filter should not be used in cases where plateau like waves are suspected (p 321) because they are basically altered by the device

Positions of the Slit

Several standard positions for the slit can be used with the patient in either the sitting or the recumbent position (Fig 85)

1 *Patient in posteroanterior position* Apex mid left ventricle and high left ventricle, pulmonary knob aortic knob high and low right atrium pulmonary veins (densogram) right and left hilar shadows (densograms) high middle and low in both the right and left lung fields (densograms)

2 *Patient in 10 degree left or right obliques* Left atrial appendage ascending aorta

3 *Patient in 45 to 60 degree left obliques* Left atrium descending aorta (densogram) high middle and low posterior ventricular border (left ventricle)

4 *Patient in 45 degree right oblique* Left atrium superior vena cava inferior vena cava (deep inspiration) upper border of diaphragm (liver tracing) high middle and low anterior ventricular border (left ventricle)

5 *Lateral positions (left lateral preferred)* Anterior ventricular border (right ventricle)

As a routine with the patient in the posteroanterior position one starts on the left side first plotting the *apex* of the heart then another tracing on the *upper part of the left ventricle* This study is followed by that of the *appendage of the left atrium* which often is better visualized by a 10 to 15 rotation toward one of the oblique positions The *pulmonary knob* is usually visible but sometimes it is advantageous to rotate slightly the patient toward the right oblique position Next the *aortic knob* corresponding to the distal portion of the aortic arch is easily studied The *descending aorta* can be studied in the left oblique position by placing the slit vertically against the spine or between this structure and the heart

On the right side one usually traces the *right atrium* at its most prominent point occasionally also at a lower point of its contour The *ascending aorta* can be studied in normal subjects by using a 10 left oblique position Its study in the posteroanterior position is possible only in mature or old individuals when atherosclerosis and dilatation of the vessels are present

It is not always possible to obtain a satisfactory tracing of the *superior vena cava* In addition to large deflections similar to those of the jugular phlebogram there are often additional vibrations due to transmitted movements On the other hand it is easy to obtain good tracings of the *inferior vena cava* in the straight posteroanterior or in the right oblique position The patient should hold his breath in deep inspiration for this procedure

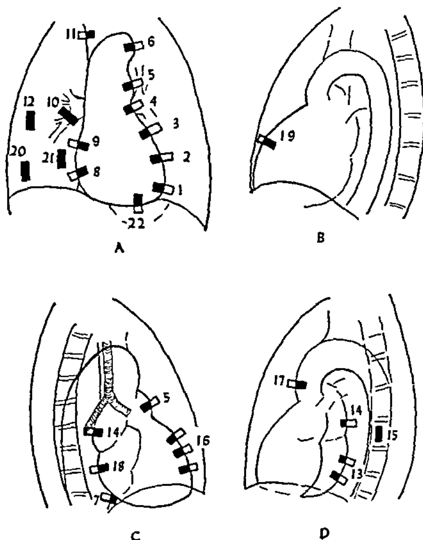


Fig 85 Locations of the pick up in various positions of the patient *A* posteroanterior, *B* left lateral *C* left oblique *D* right oblique *1* Apex *2 3* Left ventricle at center and at upper portion of left margin *4* Left atrial appendage, *5* Pulmonary arch *6* Aortic arch *7* Inferior cava *8 9* Right atrium *10* Right hilum *11* Superior cava *12* Right lung *13*, Various levels on posterior aspect of left ventricle *14* Left atrium *15* Descending aorta *16* Various levels on anterior aspect of left ventricle *17* Ascending aorta *18* Posterior aspect of right atrium *19* Right ventricle *20* Visible base of right lung *21*, Pulmonary veins *22* Lower part of left ventricle (gas in fundus) (The positions of the slit for the left hilum and the left lung are not sketched They are symmetrical with those of the right hilum and lung)

For the plotting of the *hilar pulsations* one should prefer the right hilar shadow as the one more clearly exposed The slit is placed vertically across the hilar vessels as far away from the vascular shadow as possible

For the recording of the *peripheral pulmonary pulsations*, the slit is placed vertically over either the upper or the lower field of a lung this tracing is a densogram

For the *left atrium* one can use a right oblique (sometimes almost a lateral) position and place the slit where the contour of the atrium is seen against the clear space of the right bronchus Whenever the left atrium is extremely dilated and its borderline is not clear a densogram of the chamber can be recorded

The pulsation of the *right ventricle* is best picked up in the straight lateral view just above the point where it separates from the anterior chest wall The best tracings are obtained with the subject in the recumbent position however patients with right ventricular hypertrophy yield good tracings in the sitting position also Actually the tracing of the right ventricle is often a densogram of this chamber

The *pulmonary veins* can be studied with the slit placed vertically about 2 cm beyond the convexity of the right atrium in the posteroanterior position[16] The veins coming from the middle and lower lobes of the right lung cross the descending arteries perpendicular and stand out within the bright band of the lower trunk bronchus In order to identify this venous pattern with certainty the following procedure should be followed the slit is placed vertically upon the border of the right atrium then in successive steps it is moved laterally across the bright cardio hilar interspace the final tracing being taken upon the hilum Comparison of the tracings with those of the left atrium reveals that the positive presystolic wave recorded over the 'venous field corresponds to the negative presystolic wave present in the left atrial tracing

In the study of the various cardiovascular structures the following data should be considered

1 *Amplitude of pulsation* This can be evaluated by comparing the amplitude of pulsation of one structure with that of another if they are recorded with the same degree of amplification (Fig 87) Calibration helps in the comparison

2 *Shape and time of various waves* They can be evaluated by the use of optimum amplification and by timing the waves of the eky with those of other records

3 *Abnormal movements* Transmitted and inherent pulsations can be differentiated by comparing a border tracing with a densogram of the same organ or those of two opposite borders

4 *Dissociation between various chambers* (dissociation between the atria

bundle branch block, a v block) This study is best accomplished by simultaneously recording the pulsations of two chambers (two electrokymographs)

5 *Cardiac output* Clinical determination of cardiac output has been tried by means of electrokymography The method advocated by Ring Balaban and Oppenheimer[27] was based upon tracings of density changes of the ventricular mass (ventricular densograms) Calibration of the device was necessary It is the impression of the author that this method is not suitable for general use and probably also not sufficiently accurate For this reason details of the method are omitted

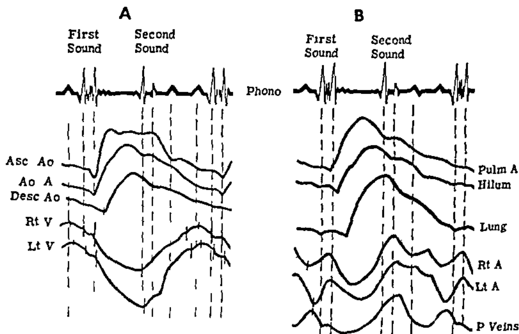


FIG 86 Schemes of various electrokymograms *A* aortic and ventricular tracings *B* pulmonic and atrial tracings

ANALYSIS OF THE NORMAL TRACING

It should be kept in mind that the electrokymogram (eky) permits the study of entirely different structures atrial ventricular arterial and venous According to the structure and its function the tracing should be compared with a physiologic or clinical tracing of atrial or ventricular contraction of arterial or venous pulsation Three main patterns can be distinguished (Figs 86 and 100)

The eky tracings represent the summation of volume changes of a chamber or vessel motions due to rotation or total shift of the heart and tractions from other structures In particular each atrium shows the effect of traction by its respective ventricle

The visible changes in volume are greatest in the left ventricle and frequently decrease in the following order (1) left ventricle with highest amplitude at the apex (2) right ventricle (3) atria (4) aorta (5) pulmonary artery (6) venae cavae and hilar shadows and (7) lungs (Fig 87)

Left Ventricle[1, 5, 19, 21, 4]

APEX Usually, a small positive wave can be recorded immediately before or during the first group of vibrations of the first sound This is due to left

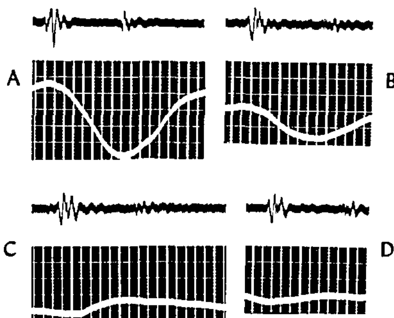


FIG 87 Comparison of four tracings recorded on the same individual with the same amplification *A* apex *B* high left ventricle *C* aortic arch and *D* pulmonic arch The phonocardiogram in *A* appears somewhat different in configuration although taken on the same subject This is due to relocation of the microphone for optimum visualization of the heart

atrial contraction which pushes a certain amount of blood into the left ventricle causing dilatation This wave is absent in patients with atrial fibrillation and large in patients with left ventricular strain The tension period is accompanied by a small depression probably due to torsion of the heart

The main ventricular wave consists of a large downward deflection which starts at the time of that large vibration of the first sound which is due to opening of the aortic valve The descending branch of this wave reaches its lowest point at a time which varies in different subjects and with various

bundle branch block, a v block) This study is best accomplished by simultaneously recording the pulsations of two chambers (two electrokymographs)

5 *Cardiac output* Clinical determination of cardiac output has been tried by means of electrokymography The method, advocated by Ring Balaban and Oppenheimer,[-7] was based upon tracings of density changes of the ventricular mass (ventricular densograms) Calibration of the device was necessary It is the impression of the author that this method is not suitable for general use and probably also not sufficiently accurate For this reason, details of the method are omitted

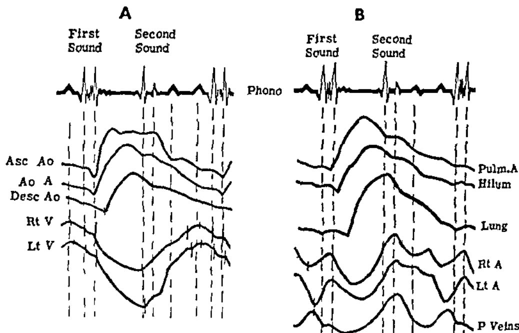


FIG 86 Schemes of various electrokymograms *A* aortic and ventricular tracings *B* pulmonic and atrial tracings

ANALYSIS OF THE NORMAL TRACING

It should be kept in mind that the electrokymogram (eky) permits the study of entirely different structures, atrial ventricular, arterial and venous According to the structure and its function the tracing should be compared with a physiologic or clinical tracing of atrial or ventricular contraction, of arterial or venous pulsation Three main patterns can be distinguished (Figs 86 and 100)

The eky tracings represent the summation of volume changes of a chamber or vessel motions due to rotation or total shift of the heart, and tractions from other structures In particular each atrium shows the effect of traction by its respective ventricle

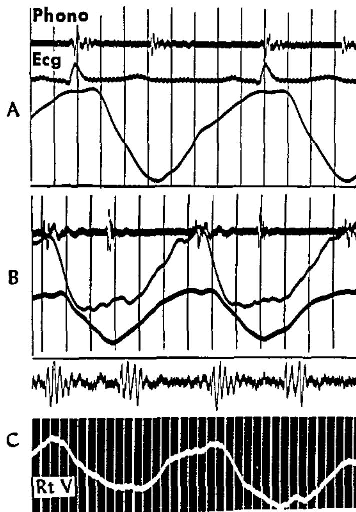


FIG 89 Ventricular tracings *A* eky record of the left ventricular border compared with phonocardiogram and electrocardiogram *B* simultaneous eky tracings of the apex and of a higher point of the left ventricular border The apex (higher eky) seems to precede the upper part (lower eky) because of more marked effect of positional factors *C* eky tracing of the right ventricle in position 19 (Fig 85)

which ends at the time of the third heart sound (phase of rapid filling) then by a more gradual slope or a horizontal line which continues until the beginning of the following cycle (Fig 88)

CONVEXITY OF THE LEFT VENTRICLE The waves reproduce volume changes of the ventricle more faithfully and denote to a lesser degree the effect of motion The coincidence between lowest point of the main wave and second sound is seen more regularly (Fig 88 89) Tracings recorded above the apex

usually seem to start later than the apical tracing This is due to less marked interference of motion phenomena and gives the impression that the contraction starts at the apex and spreads toward the base This different timing of the waves gave rise to the erroneous concept of "ventricular peristalsis when observed by roentgenkymography (Fig 89B)

OTHER POINTS ON THE VENTRICULAR SURFACE *The left ventricle can be studied in various projections, such as the left anterior oblique (posterior aspect) and the right anterior oblique (anterolateral aspect)* Tracings recorded in these positions present the same type of waves as the left margin in the posteroanterior position except that they are smaller, the lowest part of *the main wave is frequently made of a shallow curve*

The densogram of the left portion of the ventricular mass resembles an apical tracing However the ascending limb of the curve (diastole) is slower and reproduces less accurately the events of the cardiac cycle

The isometric relaxation period lies between the lowest point of the ventricular wave (if this coincides with the second sound) and the beginning of the rebound in early diastole, [2] not between that point and the peak of the rebound, as previously stated [6] The duration of this period was found ranging between 0 04 and 0 07 second by the author, 0 07 to 0 14 by Schwedel *et al* [4] and 0 02 to 0 14 by Katz *et al* [1]

Right Ventricle

Indirect evidence of right ventricular activity may be found in tracings of the right atrium in the posteroanterior view, however the data cannot be considered accurate because of influence from venous return The best tracings are recorded in the lateral views with the slit placed where the cardiac shadow separates from that of the anterior chest wall, or just below this spot *The tracing of the right ventricle is largely a densogram This tracing presents* a small positive wave at the beginning of the first sound and shows otherwise a curve which is similar to that of the left ventricle The absolute amplitude of the right ventricular wave is far less than that of the left but this may not be noted on account of higher amplification except in comparative studies (Fig 87C)

Simultaneous tracings of the two ventricles can be taken with the subject in a lateral position and two pick up units one having the slit across the anterior border (right ventricle) the other across the posterior border (left ventricle) With this technic the author found that right ventricular contraction precedes the left by 0 02–0 03 second [16] Data of Schwedel *et al* [4] show that (1) the pulmonary valve opens about 0 01 second before the aortic (possible variations 0 03 before to 0 02 after) (2) systolic ejection follows immediately the opening of the respective valve and (3) the tricuspid valve opens 0 025 second before the mitral

Left Atrium

The left atrium can be studied in three positions (1) in a 10° left oblique (left atrial appendage), (2) in left oblique at 45 or more degrees and (3) in right oblique at 45 or more degrees The three tracings are similar but one of them is frequently preferable because of individual conditions

The typical tracing (Fig 90) shows a rapid downward wave in presystole starting about 0 14 second before the first sound If the heart rate is rapid there may be only one slow wave in diastole with a peak at the end of left atrial con traction This peak usually coin cides with the first vibration of the first sound or takes place slightly before If a fourth (atrial) sound is present it occurs during the first (downward) branch of the atrial wave The presystolic wave is deeper in patients with left atrial hypertrophy and disappears in patients with atrial fibrillation After this wave the tracing rises sharply to a small positive notch during the first part of the first sound After this two negative waves are present one in systole and the other in diastole The sys tolic collapse of the left atrium is related to the dynamics of the left ventricle The left ventricular con traction lowers the a v septum and creates a suction within the atrial cavity which is compensated by increased flow of blood only gradually There fore an inward movement of the free atrial wall takes place The highest level of the tracing is reached slightly after the end of systole when the mitral valve opens After this positive notch (v wave) there is a diastolic collapse which is probably due to passive flow of blood into the left ventricle after the opening of the mitral valve The rise of the tracing during ventricular systole is steeper with the patient in the supine position

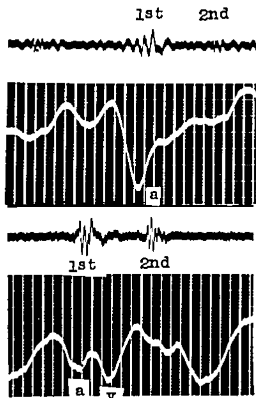


FIG 90 Eky Border tracings of the left atrium in left oblique in two normal subjects in sitting position *a* atrial wave in presystole *v* ventricular wave in early systole

When the dorsal contour of the left atrium is not visible a densogram can

be taken This presents a clear cut presystolic downward wave but is not as informative as a border tracing because of possible superimposed pulsations of the pulmonary veins and arteries The tracing of the *left atrial appendage* is sometimes not accurate during ventricular systole if the pulmonary artery is dilated, the record taken in the left oblique position may not be accurate during ventricular systole if the descending aorta is enlarged

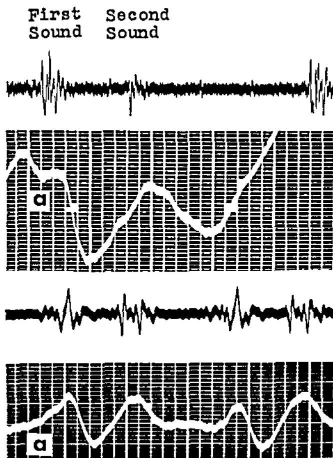


FIG 91 Eky Border tracings of the right atrium Posteroanterior two normal subjects in sitting position *a* atrial wave in presystole

Right Atrium

The tracing recorded over the margin of the right atrium is similar to that of the left (Fig 91) Contraction of the atrium during presystole is manifested by a small and rounded downward wave After this, the tracing either reaches the base line or rises above it but drops again during ventricular systole The latter is manifested by a sharp downward wave (systolic collapse) which is

usually deeper than the atrial wave The subsequent course of the atrial tracing varies with the position of the subject In the sitting position the tracing rises slowly and attains its maximum height at the time of tricuspid opening in the recumbent position the rise is quicker and there may be a convex line which brings the tracing above the base line (Fig 91) Another drop however takes place after the opening of the tricuspid valve (diastolic collapse)

In summary there is a presystolic collapse a systolic collapse and frequently an early diastolic collapse The early diastolic and the presystolic collapses are due to changes in volume of the atrium because of passive inflow into the right ventricle and because of right atrial contraction The systolic collapse is partly due to decreased atrial pressure (traction on the a v septum) and partly to total atrial displacement by the right ventricle

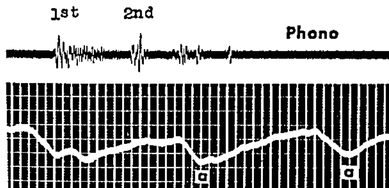


FIG 92 Eky Border tracing of the right atrium in a case of a v block showing the pattern of atrial contraction without interference from the ventricle *a* atrial contractions

The tracing of the right atrium has a superficial resemblance with that of systemic veins (Fig 93) However the (a) wave is negative instead of positive and the junction between this wave and the systolic collapse occurs before the start of ejection Therefore naming the waves with the symbols *a* *c* and *v* would be erroneous At most one could mark (a) the downward presystolic wave (v) the upward early diastolic wave In cases with a v block a pure atrial tracing is recorded (Fig 92)

Densograms of the right atrium are not always clear due to superimposition of the right atrial shadow over that of the right ventricle

Studies of the time relationship between the contractions of the right and left atrium can be made with the subject in the right oblique position Two slits are placed one above the other The higher records the left atrial contraction the lower the right With this method the author[16] found a delay of

0 025–0 03 second of the left over the right atrial contraction, this was confirmed by Schwedel *et al* [24] (Fig 93)

Aorta

ASCENDING AORTA The tracing of the ascending aorta presents a typical pattern different from that recorded over the aortic arch (Fig 94) It has an early systolic drop, a rapid rise an early peak, a slight descent (or none at

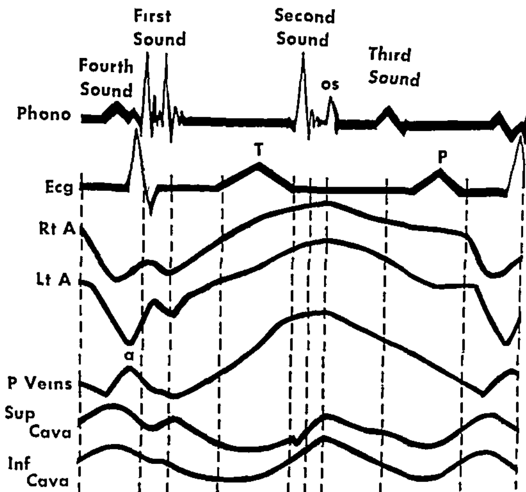


FIG 93 Scheme of atrial and venous electrokymograms

all) during the second half of systole a small incisura a high and occasionally prolonged wave after the incisura (Figs 94, 95) This tracing differs from the reconstruction of aortic pressures by Hamilton and Dow, indicating that the pattern recorded is not merely caused by volume changes (parallel to pressure changes) but is also markedly affected by motions of the heart and vessels The lowering of the aortic root by ventricular systole and the medial displacement of the ascending aorta by rotation of the heart in the same phase

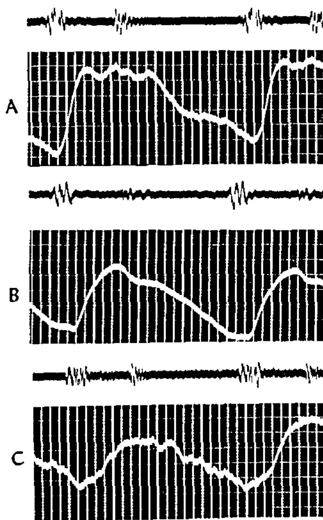


FIG 94 Eky tracings *A* border tracing of the ascending aorta (slight rotation toward left oblique) *B* border tracing of the aortic arch (left oblique) *C* densogram of the descending aorta (left oblique)

apparently reduce the height of the aortic wave This is confirmed by the initial drop of the tracing Opposite movements taking place in diastole add their effect to that of the dicrotic wave and create a high wave on the tracing The proximity of the ascending aorta to the left ventricle may contribute to the fact that, in some subjects the profile of the aortic pulse during systole greatly resembles a tracing of intraventricular pressure (Fig 100)

AORTIC ARCH The tracing of the aortic arch presents marked individual variations It has (1) a small positive wave during the first part of the first sound probably due to a rising of the aortic valves during isometric con

traction, (2) a sharp rise, after the second large vibration of the first sound (opening of the aortic valve), (3) an anacrotic depression in the first part of systole, (4) a peak during the last part of systole but well before the second sound, (5) a predicrotic notch, which may coincide with, or last slightly beyond the second sound, (6) a dicrotic wave which usually is small and *rounded and (7) a few small aftervibrations (Figs 94 95)*

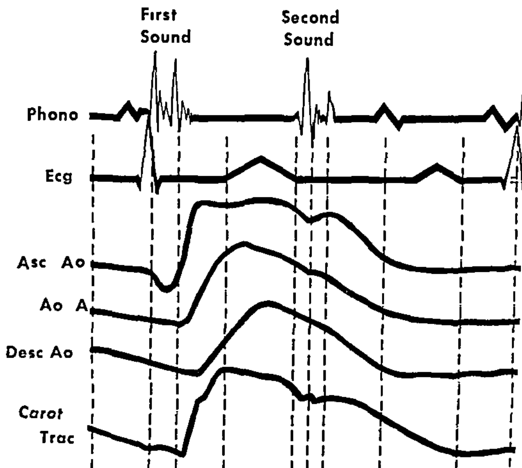


FIG 95 Scheme of aortic electrokymograms Comparison with sound and carotid tracing and with electrocardiogram

A densogram of the aortic arch gives a similar tracing However, it has been proven that the densogram and the border tracing are not identical This has been explained by pointing out that the border tracing has an important component due to lateral motion of the aortic arch

DESCENDING AORTA Since the descending aorta does not present a sharp contour on fluoroscopy, only a densogram is possible in normal subjects The tracing is similar to that of the aortic arch but shows a slight delay in the rise of the pulse in comparison with the rise in the arch (Fig 94 C)

Pulmonary Artery

The tracing of the pulmonary arch is usually easily obtained However a large left hilar shadow or a dilated descending aorta may modify the tracing The pulsations of these structures (recorded as densograms) are usually of a smaller amplitude and their influence consists mainly in a smoothing of the waves without other distortion The pulmonic pulsation starts with the open ing of the pulmonic valve (second part of the first sound) then rises sharply and occasionally shows a slight change of slope indicating an anacrotic de pression The peak is reached at about two thirds of ventricular systole The predicrotic notch is usually deep and occurs 0 06 and 0 08 second after the main vibration of the second sound The dicrotic wave is usually well defined and is higher than that of the aorta Its peak is usually 0 10–0 12 second after the main vibration of the second sound Another positive wave may be seen in late diastole before atrial contraction (Fig 96)

A densogram of the pulmonary arch is easily recorded The tracing is similar to that recorded with the slit upon the border of the vessel It may be necessary to record this densogram whenever the contour of the pulmonary artery is obscured by hilar shadows or pulmonary consolidation

In a comparative study of the aortic and pulmonic tracings by simultaneous records as well as by recording each of them with the subclavian pulse the author found a precession of 0 02–0 03 second in the rise of the pulmonic pulse over that of the pulse of the aortic arch (Fig 97) Schwedel *et al* [4] found that the pulmonic valve opens 0 01 second before the aortic valve However individual and respiratory vibrations may include from 0 03 pre cession to 0 02 delay As the progress of the pulses from the valves to the knobs varies according to elasticity of the walls and pressure of the vessels the variations found by Schwedel[24] and by Chamberlain and co workers[4] may be due to these peripheral factors

Hilar Shadows

The tracings of the hilar shadows are densograms and represent the vari ations of opacity of the hilar regions caused by changes in the blood content The amplitude of the normal hilar pulsation is between one half and two thirds of that of the pulmonary arch Additional pulsatory phenomena trans mitted from the heart and great vessels may influence the tracing without de creasing its value Following a small negative wave a large positive wave occurs (Fig 96 B) This starts approximately 0 04 second after the pulse of the pulmonary artery and 0 12 second after the beginning of the first sound The peak of the pulse wave is reached at or about the time of the main vibra tion of the second sound It may be followed by a small notch and by a small dicrotic wave The initial negative wave is synchronous with the peak of the

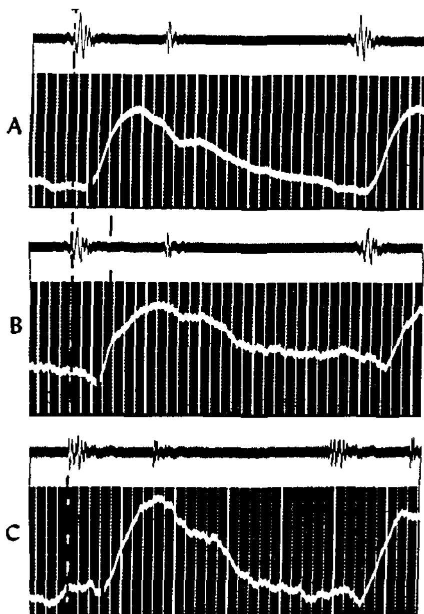


Fig 96 Electrokymograms of the lesser circulation recorded at a few seconds of interval *A*, *border tracing of the pulmonary* arch *B*, densogram of the right hilum *C* densogram of the visible base of the right lung

carotid pulse A second small negative wave is sometimes present in presystole

The positive wave of the hilar pulse indicates the arrival of the arterial pulse into the branches of the pulmonary artery However the pulsations of the pulmonary veins also influence the tracing and the early systolic depression may be due to acceleration of the venous flow

Pulmonary Veins

Tracings recorded in the right intercardiohilar space as suggested by Marchal [23] show close relationship with those of the atria In the typical tracings a presystolic positive wave (increased volume) can be noted, this is

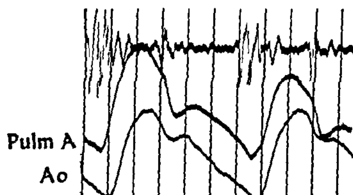


FIG 97 Simultaneous electrokymograms (border tracings) of the pulmonic and aortic arches in the posteroanterior position showing a slight precession of the pulmonic (*Pulm A*) over the aortic (*Ao*) pulse

synchronous with the negative wave (contraction = decreased volume) of the left atrial tracing Later on a systolic collapse a positive peak at the time of mitral valve opening and a diastolic collapse are present In other words the tracing is typically venous (Fig 98)

Lungs

The densogram of the lung is a tracing which resembles that of the hilum However the following differences are present (1) There is a greater delay in the rise of the pulse wave this takes place from 0 16 to 0 18 second after the beginning of the first sound and about 0 04 second after the rise of the hilar pulse (2) There may be a greater delay of the peak this occurring from 0 08 to 0 10 second after the main vibration of the second sound (3) The *curve is more rounded and exhibits no evidence of a dicrotic wave* Both the presystolic and the early systolic downward waves already noted in the hilar tracing are often present in the tracing of the lung As both arterial and

venous changes of the blood content of the lung are recorded it is possible that some of the waves as well as the peak are influenced by the effect of left atrial and left ventricular contractions, transmitted through the veins

The normal pulsation of the lung is usually about one half of that of the hilum However, there are individual variations, differences between lobes possible influence of the position of apnea or respiration (inspiration = greater waves and vice versa)

VELOCITY OF THE ARTERIAL WAVES IN THE PULMONARY CIRCULATION The velocity of the pulse waves in the lesser circulation can be studied by comparing the tracings obtained with the slit over the pulmonary knob the right hilar shadow and the visible base of the right lung using as timer the main vibration of the first sound (Fig 96)

The average times of arrival of the pulse waves in ten normal subjects were found[16] as follows 0 08 second for the pulmonary knob 0 12 second for the right hilum and 0 16 second for the base of the right lung These data were obtained by measuring on the tracing the distance from the beginning of the first sound to the rise of the wave Corrected figures are about 0 08 second lower because the opening of the pulmonic valve takes place later than the beginning of the sound

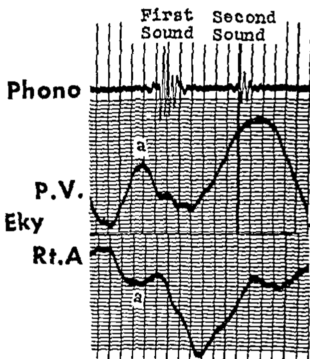


FIG 98 Electrokymogram of the pulmonary veins compared with that of the right atrium The former has a positive wave in presystole (*a*) while the latter has a negative presystolic wave (*a*) Superimposed tracings

A velocity of the pulse waves of 2 M per second between pulmonary knob and right hilum, and 2 75 M per second between right hilum and visible base of the right lung was calculated [16] While the speed of the pulse in the lesser circulation is less than in the greater being roughly one third of the latter the pulse increases its speed in the small less extensible arterioles both in the lesser and in the greater circulation

Venae Cavae

SUPERIOR CAVA A good tracing of this structure is seldom recorded in normal individuals in either the sitting or the recumbent position Occasionally, it is possible to obtain a record which resembles the jugular tracing and which shows the three typical positive waves (a) (c) and (v) Smaller vibrations are frequently superimposed (Fig 99A)

INFERIOR CAVA This tracing is recorded with the subject holding his breath in deep inspiration The best tracing is obtained with a slight rotation toward the right oblique There is a small presystolic positive wave, due to slower

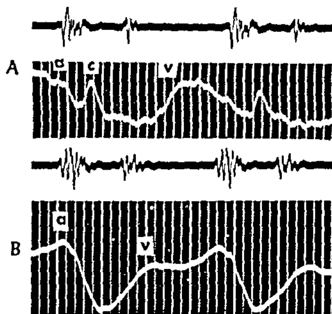


FIG 99 Electrokymograms of the superior cava (*A*) and of the inferior cava (*B*)

flow of blood at the time of atrial contraction (a wave) This is followed by a deep negative wave (systolic collapse) Then follows a slow rise culminating in a single or double wave about 0 10 second after the second sound ("v wave) This is due to slow engorgement of the vein until the tricuspid valve opens The subsequent drop reaches its maximum after the middle of diastole (diastolic collapse) No c wave is present (Fig 99B)

The tracing of the inferior vena cava is similar to the hepatic tracing of normal subjects (p 106) and is the result of the same physiologic phenomena (Figs 100 101)

PATTERN of EXPANSION

Aortic Arch
Pulmonary Arch
Hila
Lungs

PATTERN of CONTRACTION

Auricles

Ventricles

PATTERN of INTRAVENTRICULAR PRESSURE

(normal)
Left Auricle (mitral regurgit.)

FIG 100 General patterns found in electrokymography



FIG 101 Low-speed electrokymogram for the study of gradual modifications of cardiac contraction *Above* carotid tracing *below* border tracing of left ventricle *A* Before *B* three minutes after intravenous injection of a digitalis glycoside

CONCLUSIONS

Electrokymographic tracings can be recorded in a wide variety of conditions Apart from the interest that they may present for studies of cardiovascular physiology the electrokymograms may have a diagnostic value in rheumatic heart disease with mitral aortic or tricuspid defects congenital heart disease (especially conditions associated with pulmonic stenosis) syphilitic heart disease, pericarditis congestive failure differential diagnosis between mediastinal tumors and aortic aneurysms disturbances of the rate and rhythm atrial or ventricular asynchronisms alternans coronary heart disease especially in order to evaluate the extent of old infarctions, and evaluation of the effect of cardiac drugs

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CHAPTER 22

Tracings of Variations of Electrical Resistance of the Chest Due to Cardiac Action

(RHEOCARDIOGRAPHY)

HISTORY

In 1932 Atzler and Lehmann[1] described their method of *dielectrography* which was based on the study of changes in capacitance of a condenser through the modifications of position filling and shape of the heart In 1940 Rosa[5] described a somewhat different method by which the patient was again placed within the electrical field of a condenser Electrical changes caused by cardiac action were transcribed through a Braun tube In 1949 Whitehorn and Perl[7] described a modification of the Atzler Lehmann method The chest of the subject was included in parallel with the circuit of a diode The changes in frequency caused by the cardiac action were transcribed by means of a frequency modulation discriminator However Schmitz and Schaefer[6] explained the dielectrogram as an integrated cardiogram of a wide chest surface with only slight evidence of volume changes In 1945 Holzer Polzer and Marko[2] described a new method called *rheocardiography* Their method was originally applied to the limbs In a subsequent modification Polzer and Schuhfried[4] used chest leads with better results Further studies were made by Bonjer and co workers[8]

TECHNIC

Rheocardiography is based on the tracing of the changes of electrical conductivity of the human body caused by cardiac action The patient is connected with a generator beating with a frequency of 16 000 oscillations per second and with a Wheatstone bridge the latter connected so that the patient forms one arm of the circuit a current of about 20 ma flows through the body The variations of voltage caused by changes in conductivity are rectified amplified and recorded by the galvanometer of an electrocardiograph The electrodes are applied over the cardiac apex and at the right shoulder

ANALYSIS OF THE WAVES

The rheocardiogram consists of triphasic slow oscillation The tracing slowly drops at the beginning of systole rises rapidly at mid systole drops more slowly during late systole and rises again in diastole Thus there is an early systolic and an early diastolic collapse and also a smaller systolic and a larger diastolic, positive wave

Interpretation of these waves is by no means complete They seem to be related to displacement of masses of blood [8] However a much more complete study is necessary before attempting an evaluation Striking changes have been noted in various clinical conditions It is possible that the method possesses clinical value and that after proper correlation it may become of current use

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CHAPTER 23

Tracings of Discharges of Radioactive Materials within the Heart

(RADIOCARDIOGRAPHY)

HISTORY

The passage of radioactive substances through the cardiac chambers has been graphically recorded with the aid of an ink writing Geiger Mueller counter Prinzmetal and his coworkers[2, 3] described the technic in 1948 Since then the method received no further clinical applications partly on account of technical difficulties Certain modifications indicated by the author and his coworkers[1] should increase the use of radiocardiography

TECHNIC

The carefully shielded tube of a *Geiger Mueller counter* is placed over the precordium This apparatus may be of the type which gives a graphic tracing by an ink writing device as used by Prinzmetal and co workers[2, 3] or it may be connected with a direct writing electrocardiograph as used by the author and his co workers[1] The curve records the concentration of a radioisotope in the structures underlying the tube as represented by the number of disintegra tions of the radioactive element per unit of time In the original description, the curve was corrected for the random bursts by taking the mean of the

counts over a half second period In the suggested modification [1] the charac teristics of the counter and of the electrocardiograph are so adjusted that a slower curve representing the mean of counts is automatically recorded

A rapidly eliminated and short living radioisotope was initially used Prinz metal used radiosodium (Na [1]) which has a half life of 14 8 hours On the

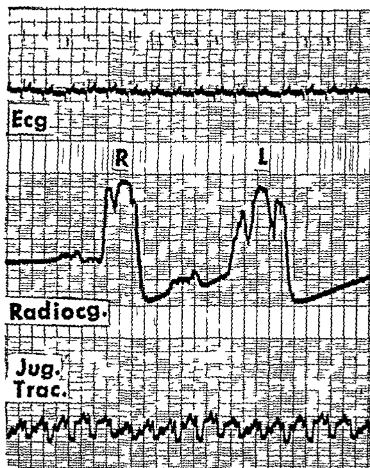


Fig 102 Rad ocardiogram in a normal young man after ad ministration of di iodofluoresceine The waves *R* and *L* ind cate pass ng of the dye through the *right and left ventricles*

other hand the author has used di iodo fluoresceine which has a half life of seven days and is eliminated in extremely minute daily amounts [1]

In order to insure rapid progress of the isotope, the extremity is either warmed before injection or exercised and the arm is raised vertically after the injection Ten to 20 millicuries of radiosodium or 50 microcuries of di iodo fluoresceine are injected into a vein of the forearm Until complete

elimination of the sodium isotope, the excretions of the patient (urine, feces) should be destroyed by special personnel according to the instructions of the Atomic Commission[4] This precaution is not necessary in the case of the di iodo fluoresceine

ANALYSIS OF WAVES

The precordial tracing consists of two main waves connected by a plateau like transitional zone The first wave is sharp starting from 0 25 to 2 seconds after the injection and lasting from 3 to 4 seconds It is due to penetration of the drug into the *right heart* and therefore is called *R* The second wave is more rounded and lower, it starts 5 to 7 seconds after the injection and lasts for 5 to 7 seconds It is due to penetration of the isotope into the *left heart* and therefore is called *L* Return of the tracing to norm indicates that all the blood containing the isotope has left the heart (Fig 102)

CONCLUSIONS

This graphic method should be useful for determination of residual blood within the heart It should give interesting data in heart failure chronic cor pulmonale coronary heart disease congenital shunts, adhesive pericarditis and obstructions to the venae cavae Slower passage of blood to or through one of the ventricles is revealed by this method Controversial problems con cerning right and left heart failure should be helped in their solution by the use of this technic

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SECTION H

Respiratory Tracings

CHAPTER 24

Tracings of External Movements Connected with Respiration

(PNEUMOGRAPHY)

HISTORY

Graphic recording of the movements of external respiration started with Marey[3] who described a device called *pneumograph* Luciani placed a cardiograph over the epigastrium and obtained good respiratory tracings Several studies were accomplished with a similar technic by Mosso[5] and Hofbauer[1]

A Frank's capsule was applied later to the same devices and used for photographic recording of respiration

For several years the author has used a linear microphone connected to a pneumatic cuff wrapped around the chest or the abdomen Thus chest or abdominal movements due to respiratory dynamics were recorded electrically

TECHNIC

A 25 cm long section of rubber tube having a diameter of 2 5 cm is closed at both ends and glued on a strip of firm cloth or leather A short piece of metal tube of 3 mm in diameter is fastened through a hole of the chamber formed by the tube so that it is airtight This device constitutes the pneumograph It is placed evenly around the abdomen or the chest and fastened by strings Expansion of the chest or abdomen causes compression of the air in the pneumograph

A Miller White crystal microphone[4] which gives a linear response (p 28[2]) is connected with the pneumograph and with the input of an electrocardiograph This microphone should be so adjusted that an increase of pressure gives a downward movement of the tracing Then inspiration is revealed by a downward movement expiration by an upward movement * Simultaneous tracings of the thoracic and abdominal respiration can be recorded with two pneumographs two linear microphones and a double channel electrocardiograph

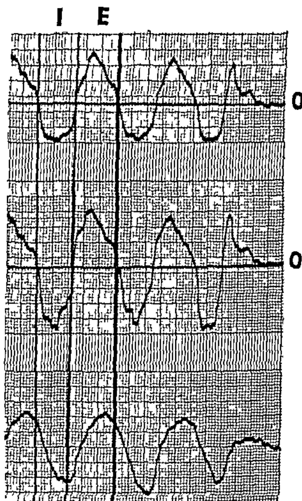


FIG 103 Pneumotachogram (above), pressure curve from the mouth (center), and low thoracic respiratory tracing recorded with three crystal microphones of a linear type

ANALYSIS OF WAVES

Respiratory dynamics is revealed by a simple monophasic V shaped wave The downward slope corresponds to inspiration and is shorter the upward slope corresponds to expiration and is longer (Fig 103) *Smaller superimposed waves are caused by* pulsations of the heart (thoracic respiration) or the aorta (abdominal respiration)

In general the curves of thoracic and abdominal respiration are parallel and simultaneous However, abdominal respiration is usually more ample in the male, *thoracic respiration* in the female Slight differences in timing may be caused by imperfect application of the pneumographs which modify slightly the tracings on account of the elasticity of the tube and the air contained Other devices like the Harvard Co pneumograph can be used If they involve a spring the causes of error are increased

* This arrangement is possible (1) if one has a crystal microphone with two air outlets *one on each side of the crystal* (2) *if one can invert the polarity of the galvanometer* If neither is possible the tracing is inverted and note of this should be taken

CONCLUSIONS

Recordings of respiratory dynamics have been made during dyspnea, and can be used for the differential diagnosis between paroxysmal cardiac dyspnea and bronchial asthma Tracings of the external movements of respiration may be preferred to those of pressure or velocity from the air passages on account of easier technic However it should be kept in mind that they are less accurate

Simultaneous thoracic and abdominal tracings are taken if dissociation between thoracic and abdominal respiration is suspected because only records of the external movements of respiration can be used to diagnose this severe neurologic disturbance

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CHAPTER 25

Tracings of Pressure and Speed of Respiration

(PNEUMOTACHOGRAPHY)

HISTORY

Following studies on graphic tracings of external respiratory motions several authors investigated the changes of lateral pressure from one of the nostrils or from the mouth during normal respiration These were recorded at first on smoked paper by means of a Marey tambour, then on a photo graphic film by means of a Frank s capsule last on the film of an electro cardiograph by a crystal microphone

The velocity of the air from the mouth was studied first by Fleisch who described a special instrument the *pneumotachograph* This based on the principle of a differential Frank's capsule recorded the tracing by means of a mirror fastened on the membrane *inside* the capsule both the direct and the reflected beams passed through the glass closing the capsule Another less perfect pneumotachograph was described by Hochrein [3] Pneumotachographic tracings of normal subjects were studied by Bretschger [1] Both the original method of Fleisch[4] and a new method based on a pneumotachograph and a differential crystal microphone * were used by the author

TECHNIC

Tracings of Pressure

A rubber tube having a lumen of 3 mm is connected with a glass or bakelite olive at one end with a linear microphone at the other The olive is introduced

* This was specially built by the Sanborn Co of Cambridge Mass

into one of the nostrils of the patient who is instructed to breathe evenly through the nose The jack of the linear microphone is connected with the input of an electrocardiograph and the tracing is recorded either photographically or by direct writing methods The zero line is found by the patient holding his breath for a few seconds During apnea irrespective of the phase of respiration in which the chest is immobilized the light beam (or the pen) traces a straight line which is the zero line If the subject has some impairment of nasal respiration a mouthpiece, like those used for the study of basal metabolism is placed into the mouth of the subject It is connected with a short open piece of tubing having a thin metal tube on one side The latter is connected with a linear microphone and the patient is instructed to breathe evenly through the mouth If needed a nose clamp is used in order to prevent nasal respiration

Tracings of Velocity

Fleisch s pneumotachograph is made as follows a series of thin tubes having a lumen of 3 mm * is contained inside a larger metal tube provided with a mouthpiece Two side tubes are connected with one of the thin tubes † *These are connected by means of short rubber tubes with the ends of a differen*tial linear microphone connected with the electrocardiograph (p 154) The proximal tube is connected with that end of the microphone which gives a positive deflection for an increase in pressure the distal tube with the other end

The mouthpiece of the pneumotachograph is placed into the patient s mouth If necessary a nose clamp is used in order to prevent nasal respiration At one time during the taking of the record the patient is invited to hold his breath The light beam or the pen of the electrocardiograph then traces a steady line which represents the zero line of the tracing

Objections may be raised against the use of the linear microphone for either velocity or pressure curves of respiration because it does not register extremely slow deflections However respiratory waves are still within the field of action of this microphone and only a minimal deformation of the curve may be expected consisting of a slightly earlier flattening of the curve at the end of each phase ‡

* The larger tube contains 99 thin tubes All of them are used whenever the patient has dyspnea only 33 of them are used for normal respiration The exclusion of the extra tubes is obtained by closing a central section of 66 tubes with a rubber stopper

† Actually there are two sets of side tubes One couple is separated by a distance of 5 cm a second couple by a distance of 10 cm The two couples of tubes were made in order to permit choice between two sets giving a different amplitude of the waves As electrical recording has its own device for increasing amplification only one set will be used constantly while the other should be closed If velocity and pressure tracings are simultaneously recorded one tube of the second set is used for the tracing of pressure while the other tube is closed

‡ Comparative tests made by the author with a linear microphone and a high sensitivity electromanometer show that the former is entirely adequate for clinical tracings

In order to avoid displacement of the base line no use of the instomatic device for automatic adjustment of the tracing should be made during the entire test

ANALYSIS OF WAVES

Both the pressure and the velocity tracing consist essentially of two rounded, diphasic waves During inspiration, the curve drops then slowly flattens and returns to the zero line Expiration is not separated from inspiration by an accident of the tracing Only the zero line artificially traced, divides the two phases During expiration, the tracing rises slowly above the zero line reaches a maximum then slowly tapers off and reaches again the zero line (Fig 103) Expiration is somewhat longer than inspiration and may become much longer in clinical cases (Fig 265) Small notches due to cardiac action are present in both phases They have been already considered (p 116) and can be studied with slight modifications of technic

As shown in Fig 103 there is but a small difference between the pressure and the velocity tracing Therefore, except in special cases the first easier type of tracing should be preferred for clinical studies

Inspiration is accompanied by a decrease of pressure in the respiratory passages this causes suction of air and a drop of both the pressure and the velocity tracings Expiration is accompanied by an increase of pressure in the respiratory passages this causes expulsion of air and a rise of both the pressure and the velocity tracings The time relationship between these waves and the external movements of respiration (p 211) is apparent in Fig 103 The end of inspiration is revealed by the lowest point of the curve of external respiration and by a return to the zero line of the curves of pressure and velocity The end of expiration is revealed by the highest point of the curve of external respiration and by the return to the zero line of the curves of pressure and velocity

CONCLUSIONS

Pneumotachography was used for various physiologic studies including one on cardiac output which have subsequently lost value Clinical tracings of pressure are accurate and present some interest in the differential diagnosis of the various forms of dyspnea

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CHAPTER 26

Tracings of Bronchial Functions

(ELECTROBRONCHOGRAPHY)

HISTORY

A series of studies on bronchial and pulmonary functions by the author led to description of a method called *electrobronchography*. These studies were first developed on experimental animals[1, 2, 3] and subsequently applied to human subjects.[4]

TECHNIC

The method is based on the use of a conventional electrocardiograph, a preamplifier, and a low pass filter. The latter consists of condensers which sharply decrease the amplitude of waves above 80 cycles per second, like the QRS complex of the electrocardiogram, but cause only small changes for waves of lower frequencies. The lead is bipolar. One electrode consists of a 50 cm. long copper wire insulated along all its course except at the tip. The latter is silver coated and tightly wrapped with cotton soaked in isotonic solution of sodium chloride. The other electrode is a round German silver electrode smeared with Redux jelly. Following novocaine anesthesia of the pharynx and glottis, the wire electrode is introduced into a small bronchus, possibly within the lower lobe of the right lung. The other electrode is applied over the trachea and held by a rubber strap wrapped around the neck. One of

the wires is connected with the filter the preamplifier and the galvanometer the other only with the preamplifier and galvanometer

Simultaneously with the electrobronchogram (ebg) an air pressure tracing is recorded from one of the nostrils (p 214) Anesthesia of the upper respiratory mucosae can be dispensed with in subjects with tracheal fistula

ANALYSIS OF WAVES

The ebg consists essentially of a double monophasic wave similar to that of the pressure tracing from the air passages The polarity of the apparatus should be adjusted so that inspiration is accompanied by a negative wave, expiration by a positive The ebg is the resultant of the action currents of the smooth fibers of the trachea and bronchi down to the smaller ramifications it is possible that additional currents are caused by physical changes connected with variations of length and caliber of the bronchi and distention of the pulmonary parenchyma Action currents of the striated muscles are excluded by this method Action currents of the heart are revealed by small complexes having a cardiac cycle

The main ebg waves have a respiratory cycle and are similar to the waves of a pressure tracing They represent mainly the electrical expression of tonic changes of the bronchial muscles which become longer and wider during respiration shorter and narrower in expiration Abnormalities of the bronchial function are revealed by changes in the height and phase of the ebg waves independent of changes of the respiratory dynamics

CONCLUSIONS

The electrobronchogram has received so far limited applications It lends itself to studies of bronchial and pulmonary functions and can be used in cases of cor pulmonale and chronic or subacute dyspnea It can be used also for pharmacologic studies

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CHAPTER 27

Tracings of Volume of Air

(SPIROGRAPHY)

HISTORY

The first spirometer was built by Hutchinson[3] in 1860 Spirometric studies by Mosso [6] Vierordt [10] and Tigerstedt[9] on animals and man gave many basic data on respiration After the initial stage spirometry was developed together with apparatus for determination of basal metabolism Thus Atwater,[1] Benedict,[2] Roth,[7] and Krogh [4] have developed both the method and the instrument

TECHNIC

Any clinical apparatus for basal metabolism can be used for spirometry In particular modern, waterless apparatus are quite practical The patient should be placed in a comfortable sitting position The mouthpiece is placed between the teeth and a nose clamp is applied The patient is invited to breathe evenly for a few minutes Then he is invited to exhale deeply, to inhale deeply and again to exhale deeply The tracing represents the spirogram Inhalation is represented by a downward movement exhalation by an upward movement of the stylus The number of cubic centimeters is directly measured in the graph

ANALYSIS OF TRACING

As known the amount of air passing in and out during ordinary respiration is called *tidal air* it averages 500 cc The amount which can be forcibly inhaled after a forced exhalation may reach 3000 cc and is called *complemental air* Forced exhalation from a position of rest may amount to 1000 cc this is the *supplemental air* Complemental air plus supplemental air represents the *vital capacity* The simplest way to determine the latter is to perform first a forced exhalation then a forced inhalation then again a forced exhalation The line which connects the highest with the lowest point represents vital capacity

CONCLUSIONS

The tracing gives information about normal tidal air and vital capacity The former is reduced whenever there is engorgement of the pulmonary vessels or pulmonary edema the latter is reduced not only by pulmonary congestion but also by limited mobility of the diaphragm or the chest wall (liver enlargement ascites pleural effusion pleurisy) The data have value only if they can be compared with previous readings on the same individual because individual variations are large

Being recorded on slow motion papers the spirograms give a visual exact evidence of certain types of respiratory disturbances The Cheyne Stokes types of periodic respiration (Fig 266) as well as the sighing respiration of neurocirculatory asthenia are revealed by this method

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SECTION I

Tracings of Electrical Phenomena of the Heart and Vessels

CHAPTER 28

General Electrocardiographic Technic

HISTORY

Animal electricity was accidentally discovered by Galvani[4] in 1791 Evidence that the cardiac muscle is the source of electrical currents was given by Matteucci[10] in 1843 and by Koelliker and Mueller[7] in 1856 The last two authors recorded these currents by means of a neuromuscular preparation (*frog gastrocnemius*) and used this *physiological rheoscope in order to* prove that the electrical manifestations of the heart precede the mechanical Subsequent studies on cardiac electricity were made by Bernstein[1] and by Engelmann[3] In 1873 Lipmann[9] described the *capillary electrometer* which soon was employed by Marey[9] for studies of animal electrocardiography and by Waller[11] for studies of human electrocardiography At the beginning of the twentieth century Schweigger described the first *string galvanometer* and soon afterwards Einthoven applied this instrument to the graphic registrations of electric currents of the heart

Einthoven s original apparatus was large and cumbersome Today, however, it is a portable instrument capable of simple operation

In Germany not many years after the first string galvanometer was perfected a rotating mirror and coil type of instrument was introduced having sufficient sensitivity for registration of cardiac potentials (*mirror galvanometer*)

With the development of the thermionic vacuum tube it became possible to register electrocardiograms by means of oscillographic units coupled with electronic amplifiers While the string galvanometer registers current flow the thermionic or electronic electrocardiograph records electrical potentials These may be amplified up to one thousand times without distortion and are recorded by a sturdy oscillometer

Two schools greatly contributed to electrocardiography in the first quarter of the twentieth century that of London with Sir Thomas Lewis as its exponent and that of Vienna led by Wenckebach and Rothberger The studies of arrhythmias and of heart blocks were practically completed in that time Between 1920 and 1930 American researchers (Pardee Smith Herrick) established the basis for the clinical recognition of coronary and myocardial lesions

Between 1930 and 1950 fundamental contributions were made by Wilson and his school Interest in the unipolar leads already described by Wilson was awakened by Goldberger s studies

APPARATUS

Modern electrocardiographs are described in detail in Appendix A Four different types of instruments have been used (1) the string galvanometer (2) the oscillograph or mirror galvanometer (3) the cathode ray electro cardiograph and (4) the oscillograph with thermionic amplification

The cathode ray electrocardiograph has several practical and technical dis advantages for present day use The oscillograph has been abandoned as such and incorporated in the thermionic tube amplifier Therefore only the string galvanometer now made portable and the amplifier electrocardiograph are in current use From the point of view of the fidelity of registration these two in struments are practically equal However lack of inertia stability of amplifica tion and greater sturdiness render the latter superior to the former

Two methods of recording are used the photographic and the direct writing The *photographic method* used in both the string and the amplifier types of galvanometer is theoretically superior because it avoids any friction with the film and is less liable to acquire imperfections Whenever simultaneous tracings of heart sounds and electrocardiograms are desired this is the method of choice The *direct writing method* avoids the need for photographic process ing of the film and is now currently used The possibility of immediate in spection of the film renders this method preferable whenever delicate multiple tracings are recorded and for bedside diagnosis The method is based on the color change of a special film caused by a heated stylus Ink writing devices are less perfect and should be used only for mass work

ACCESSORIES AND CONTROLS

Although the various electrocardiographs differ in appearance and arrangement of controls, several accessories are common to all (1) sensitivity control (2) centering device, (3) lead selector, (4) lead marker, (5) calibration device, (6) cables and electrodes (7) button for instantaneous automatic block of the pen or light beam, direct writers with a hot stylus have also (8) a device for regulating the temperature of the stylus

Calibration

Evaluation and comparison of electrocardiograms (ecg s) necessitates the knowledge that a certain height of deflection corresponds to a certain differ

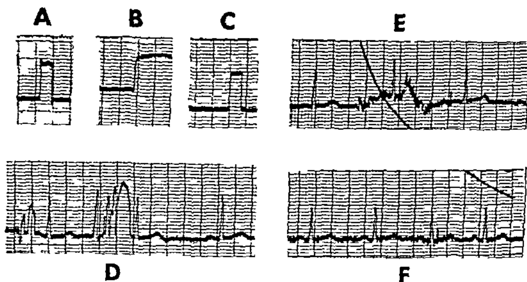


FIG 104 Possible artifacts of an electrocardiogram *A*, overshooting *B* over damping *C* normal *D* sudden jerks due to improper contact of the electrodes *E*, movement of the patient *F*, muscular tremor

ence of potential Therefore all apparatus are provided with a calibration device This consists of a button which introduces a current of one millivolt into or excludes a resistance of one millivolt from the circuit The button is depresed while the film is moving and the sensitivity is adjusted until a positive deflection of one centimeter is recorded

The button for calibration (or standardization) is also used in order to check the characteristics of the apparatus The angles of the rectangle should be square with minimal evidence of rounding (Fig 104C) Otherwise an incorrect picture of the various waves would be obtained If the button is depressed and the finger is not lifted the displacement of the beam persists *in* the string galvanometer, in the amplifier type of apparatus, the beam returns

slowly to the baseline This return should be slow reaching two thirds of the deflection in 1 5 second The frequency period of a good electrocardiograph is such that under critically damped conditions 80 per cent of a final deflection should be reached within 0 01 second

The *centering device* controls the position of the beam and is used in order to regulate the baseline of the tracing Usually the baseline is placed near the center of the film however if large monophasic deflections are present it may be convenient to move it up or down in order to avoid distortion or non registration of part of the waves *

The *lead selector* is marked for standard and for the various leads Present apparatus have first the positions for the conventional limb leads then those for the unipolar limb leads (see p 231) It is likely that in the future the opposite will be true or that the standard leads shall be abandoned

Lead Marker

This gives dots or lines on the film In the author s laboratory the following marks are used

1 R or V1 =	V4 = -	V7 = -	VE = --
2 L or V2 =	V5 = -	V8 = -	
3 F or V3 =	V6 = -	V9 = --	

Cables and Electrodes

The power cord connects the instrument with the power except in the cardiographs working with batteries like the Sanborn Stetho Cardiette The patient cable is divided into three four five or more wires The ends of the wires are thus marked *RA* = right arm *LA* = left arm *RL* = right leg † *LL* = left leg *C* = chest (or *B C D* = various chest electrodes)

The normal limb electrodes are 3 × 5 cm rectangles of German silver The chest electrodes made of the same alloy are round and have a diameter of 3 cm ‡ Both have a small conic knob or small teeth directed upwards used for fastening to the limb by means of rubber straps The chest electrodes can be held by a long rubber strap or by a bakelite handle

Ground Wire

Most apparatus and especially those working on AC require grounding for this purpose either a special cable or an ordinary copper wire with two clips can be used One of the clips will be attached to a metal pipe available in the room (faucet radiator etc) or to a special ground wire

* If the deflections are too large and diphasic one can reduce the degree of sensitivity of the apparatus to a fraction of the normal value indicating this by the symbol $\frac{N}{X}$ (e g $\frac{N}{2}$)

† This wire is used only in order to avoid AC interference

‡ In infants and young children a chest electrode of 1 5 cm in diameter should be used

PREPARATION OF THE PATIENT

A supine or semirecumbent position is preferred because it insures complete muscular relaxation and avoids superimposition of currents of skeletal muscles on those of the heart (Fig 104F) A long wide and comfortable cot with adjustable back can be used If the patient has orthopnea, he should be propped up with pillows If the tracing is taken in the sitting position a large arm chair should be used The feet of the patient should be raised from the floor and rest on a nonmetallic low stool It may be necessary to shave the chest and limbs of a male patient if there is excess of hair In general however a generous supply of electrode jelly* avoids this need

Patients with somatic tremor represent a technical problem because the muscular hypertonus or clonus is the cause of artifact (Fig 104E F) It has been suggested[6] to replace the limb electrodes by four round electrodes similar to those used for the chest leads, these should be attached with scotch tape or adhesive over the supraglenoid tubercles and the anterior superior iliac spines where tissue movement is minimal

Amputees can be studied by applying one of the electrodes over the stump of the missing limb

The patient s skin is gently rubbed with electrode jelly which insures a good conductivity The jelly may be rubbed with a toothbrush or with the edge of the electrode Then the electrodes are applied

FILM

Photographic film has the time marked by vertical interruptions of the light beam caused by the spokes of a turning wheel Direct writing films have a marking on the paper which corresponds to the conventional speed of 25 mm per second Each thin line represents 0 04 second, each thicker line 0 2 second, every fifth thick line a second Even though the photographic film may move at different speeds the time marking has always the same value On the other hand direct writing films have already marked time lines if they move at faster or lower speeds the lines acquire a different value if the film moves at 50 mm per second, each thin line represents 0 02 second and each thick line 0 1 second

Both the photographic and the direct writing film have horizontal lines at 1 mm intervals They are used for measuring the height of the various deflections Each millimeter represents 0 1 millivolt in tracings recorded with conventional amplification

* This contains sodium chloride or zinc sulfate pumice powder and glycerine

ELECTROCARDIOGRAPHIC ROOM

The tracing may be taken anywhere However a special room is usually assigned to electrocardiography in hospitals and laboratories This room should be as far away as possible from roentgen ray short wave and other electrical equipment which might be used during the taking of the tracing It should be as far away as possible from high tension cables and electric motors like those of elevators and street cars In spite of these theoretical considerations good tracings are frequently taken in roentgen ray rooms if all possible apparatus is disconnected

Whenever simultaneous roentgen ray and electrocardiographic tracings are needed (as in the case of electrokymograms and electrocardiograms) a special filter interposed between patient and electrocardiograph is necessary In other cases electrical interference can be avoided by screening the bed and the patient with wire mesh nets A single wire is then used for grounding the nets

ARTIFACTS

The possible artifacts are many but they can be easily discovered and eliminated or discounted They are (Fig 104)

1 AC interference from improper isolation of the room or patient
2 Muscular currents caused by somatic tremor
3 Sudden jerks of the pen or beam on account of technical faults of the instrument or improper application of the electrodes
4 Slow wandering of the baseline caused by changes of conductivity of the skin (sweating vascular changes) or respiration (chiefly in the chest leads)
5 Abnormal direction of the waves caused by wrong connection of the cables
6 Improper shape of the waves due to either overshooting or overdamping of the apparatus

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CHAPTER 29

The Electrocardiographic Leads and the Electrocardiogram

It is well known that during muscular contraction the part of the muscle which is stimulated becomes negative in relation to the rest If a muscular fiber A B is stimulated in A this point becomes negative in relation to B If an electrode is placed above the point which is stimulated and another at a distance the galvanometer records a negative deflection at the time of stimulation If two electrodes are placed at the opposite sides of a point stimulated no current is recorded by the galvanometer the two electrodes are at equipotential points

Bipolar lead is the name given to any lead using two electrodes placed at the opposite sides of and at some distance from the source of electric currents (heart) The record represents the algebraic summation of two potentials of similar values and frequently with opposite polarity Examples are the old standard leads

Semi unipolar lead is the name given to any lead using one electrode near the source of the electric currents (heart) while the other is at a greater distance The record again represents the algebraic summation of two potentials one of them is of a high value because recorded near the source the other has a smaller value because recorded at a greater distance from the source of currents Examples are the old Lead IV (anteroposterior) and the various CF and CR leads

Unipolar lead is the name given to that lead using one electrode near the source of electric currents (heart) while the other is in an area of zero potential (e g at an 'infinite distance' from the source of currents) The record represents the expression of the 'absolute' changes of potential in the area lying under the electrode near the source of currents (heart) because it registers the difference in potential between that point and zero The concept of a 'zero potential electrode' is extremely valuable but difficult to realize The best solution seems to be the "central terminal" of Wilson (p 231) In

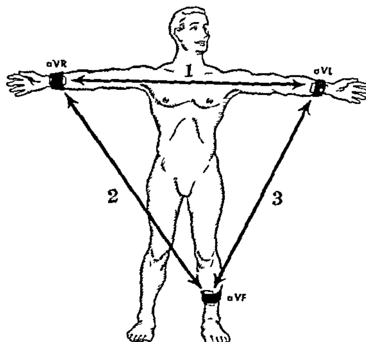


Fig 105 Sketch of the unipolar and bipolar (standard) limb leads

both the semi unipolar and the unipolar leads, the electrode placed near the heart is called the *exploring electrode* while the other is called respectively *semi indifferent* or *indifferent*

STANDARD LIMB LEADS

The standard limb leads were the only ones used from 1905 to 1935 and are still employed These *bipolar limb leads* are the following (Fig 105)

Lead 1 connecting the right with the left arm
Lead 2 connecting the right arm with the left leg
Lead 3 connecting the left arm with the left leg

It is apparent that each of the bipolar limb leads represents the algebraic summation of the potentials of two limbs The following formulas explain the relationship between the three unipolar and the three bipolar (standard) limb leads

$$V_R+V_L+V_F = 0 \quad V_L+V_R = -V_F$$
$$1=(V_L - V_R)K$$
$$2=(V_F - V_R)K \quad 2 = 1+3$$
$$3=(V_F - V_L)K$$

K is a coefficient which varies according to calibration and technic

Lead 1 reports the variations of potential of the left lateral cardiac wall (right or left ventricle according to the position of the heart but mostly left ventricle) minus those of the atrial and ventricular cavities Lead 2 reports the variations of potential of the diaphragmatic surface of the heart minus those of the atrial and ventricular cavities Lead 3 reports the variations of potential of the diaphragmatic surface of the heart minus those of the left lateral cardiac wall Certain anterior and posterior regions of the heart are the source of potentials which are not pictured by standard leads These regions have been called *silent areas*

The standard limb leads are still used because most of the known clinical patterns were recognized through them In spite of their traditional use it is likely that in the future they shall be abandoned

UNIPOLAR LIMB LEADS

Following Wilson s suggestion[10] it is possible to study the variations of potential of any point of the body without interference from currents of other points In order to do this the three limbs are connected together by three wires and three resistances of 5000 ohms or more To this central point of connection called the *central terminal* is attached one of the cables of the electrocardiograph Based on the principle of Einthoven s triangle the central terminal is supposed to have zero potential and to act as an indifferent electrode * The other cable of the electrocardiograph is connected with any point of the body by means of an electrode called the *exploring electrode* In the case of the limb leads the exploring electrode is connected successively with the right arm the left arm and the left leg The three tracings are called *VR VL* and *VF* (Fig 105)

Goldberger suggested eliminating the electrical resistances of the central terminal and disconnecting the wire of the limb under study in order to increase the amplitude of the waves The Goldberger leads are called *augmented unipolar leads* and their symbols are *aVR aVL* and *aVF* Successive studies[13] have confirmed that disconnecting the wire of the extremity

* Actually the central terminal has small changes of potential which can be disregarded in clinical tracings because they do not exceed 0 1 mv

under study does not change the shape and polarity of the waves while increasing their magnitude On the other hand, the presence of the resistances is necessary in order to have a zero potential' central terminal Present day technic therefore embodies the best features of the Wilson terminal but with alternate disconnection of the wire of the terminal from the limb under study For simplicity s sake, the symbols of the three limb leads with this technic can be called *R*, *L*, and *F*

Connection of the three wires with the central terminal and disconnection of the wire of the limb under study is obtained automatically in most modern apparatus where the lead selector is successively placed in positions aVR, aVL and aVF

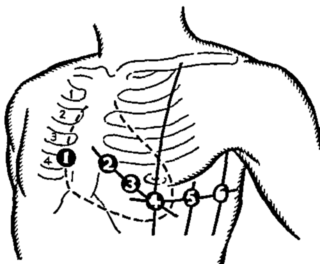


Fig 106 Locations of the chest electrodes for leads V1 to V6 The heart has been drawn enlarged in order to show that point 4 may not coincide with the apex

CHEST LEADS

Semi unipolar chest leads have been taken for several years by placing one electrode on the chest while the galvanometer was connected with the electrode of any of the limbs The symbols used were Chest right arm = CR leads, Chest left arm = CL leads Chest left leg = CF leads These leads now have only historical interest because the tracings obtained by their use represented a summation of the potentials of the chest plus those of one of the limbs

The unipolar chest leads are now generally accepted They are taken by placing a round exploring electrode on the chest while the galvanometer is connected with Wilson s central terminal (indifferent electrode) The symbols used are CV leads, or simply V leads

Following the recommendations of the Committees of the American Heart Association and the Cardiological Society of Great Britain and Ireland [1] six chest locations (Leads V1 to V6) are commonly used (Fig 106) * To these, several others have been added for use in special cases

* There has been controversy on the number of chest leads that should be taken if one wishes to skip some of the six leads usually included in the routine use If the electrocardiogram is taken by a technician at least four routine chest leads are necessary (V1 V2 V4 V5) If it is taken by a physician as in the author s laboratory three chest leads (V2 V4 V6) may be

1 Three posterior locations [9] (Fig 107) for the study of high posterior potentials of the heart (Leads V7 V8, V9)

2 One xiphoid or ensiform location [9] (Fig 108) for the study of postero diaphragmatic potentials (VE) †

3 Three high anterolateral locations[15] (Fig 108) for the study of the high anterolateral potentials (high V4 high V5 high V6)

4 One high right sternal lead (Fig 108) for the study of right atrial potentials (high V1)

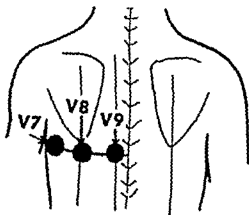


Fig 107 The posterior locations for chest leads V7 to V9

5 Three right chest leads[14] for the study of potentials of the right heart in cases with right heart enlargement (V3R V4R V5R) ‡

6 Four esophageal leads (p 247) for the study of posteroinferior potentials (left atrial and ventricular) (Fig 116)

Locations and symbols of 16 leads are described in Table 6

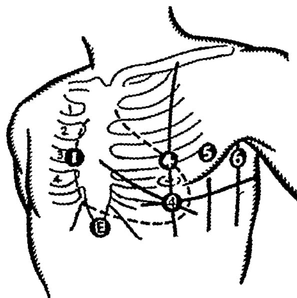


Fig 108 Special electrode locations High V1 high V4 high V5 high V6 conventional V4 VE

sufficient. It is understood that in any case where there is suspicion of coronary heart disease all the routine leads V1 to V6 should be taken and that whenever a myocardial infarct is suspected additional special leads should be employed In infants V1 V3R and V4R should be taken systematically

† According to Soulie and Laham [16] a lead VE2 may be superior to VE To obtain this the electrode is placed over the sixth space at the left of the xiphoid on the prolongation of the left marginal line of the sternum This lead has normally positive P and T waves and an rS pattern

‡ V1 is the topographic equivalent of V2 but on the right chest V3R is the equivalent of V3 but on the right chest. V4R is the equivalent of V4 but on the right chest.

TABLE 6 LOCATIONS AND SYMBOLS OF ELECTROCARDIOGRAPHIC LEADS

No	*Exploring Electrode*	*Symbol*	*Use*
1	Fourth intercostal space near *right* sternal margin	V1	Routine
2	Fourth intercostal space near *left* sternal margin	V2	Routine
3	Midway between V2 and V4	V3	Routine
4	Fifth intercostal space midclavicular line	V4	Routine
5	Third intercostal space midclavicular line	High V4	Special
6	Same level as V4 anterior axillary line	V5	Routine
7	Third or fourth space anterior axillary line	High V5	Special
8	Same level as V4 mid axillary line	V6	Routine
9	As high as possible under axilla mid axillary line	High V6	Special
10	Same level as V4 posterior axillary line	V7	Special
11	Same level as V4 mid scapular line	V8	Special
12	Same level as V4 paravertebral line	V9	Special
13	Tip of ensiform process	VE	Special
14	Third right interspace near sternum	High V1	Special
15	Fourth intercostal space within right mid clavicular line	V3R	Special
16	Fifth intercostal space right mid-clavicular line	V4R	Special

ANALYSIS OF WAVES

General Analysis

The electrocardiogram (ecg) is a composite curve in which various accidents or waves have a definite position These waves are called *P Q R S* and *T* The first part of the ecg composed of the *P* wave and of minor accidents is called the *atrial* (or *auricular*) *complex* because it bears a definite relation to the activity of the atria (auricles) The second part of the ecg composed of an initial rapid complex (QRS) and a slow final deflection (T), is called the *ventricular complex* because it is connected with the activity of the ventricles (Figs 7, 109 and 110)

The excitation of the s a node is not recorded in clinical tracings but can be recorded by means of an intraatrial electrode As there is no way of detecting it in the routine cases it is assumed that it precedes the beginning of P by a small fraction of a second (0 05 sec) The P wave is the resultant of the activation or depolarization of both atria It is a blunt deflection lasting from 0 08 to 0 10 second in normal individuals Whenever transmission of the impulse to the left atrium is impaired or the wall of the left atrium is distended or hypertrophied the P wave shows a double peak or a diphasic configuration (Fig 220) In such cases the first or upward phase of P is due to the right atrium, the second or downward, phase of P is due to the left This is frequently more apparent in leads V1, aVF and 3 Inactivation or repolarization

of the atria is usually not revealed by the ecg However cases with prolonged a v conduction or a v block may show a small dip called the atrial T wave (*Ta wave*) due to repolarization of the atrial myocardium (Fig 117)

The P wave is normally followed by a period during which the electrocardiogram reveals no deflection The stimulus starts to spread at the beginning of P and reaches the a v node during P activation of the a v node and diffusion along the bundle of His takes place between P and Q The interval between the initiation of P and that of QRS (called *P R interval*) measures the sum of (1) sino nodal and sino atrial conductions (2) nodal latency and (3) nodo septal conduction Normal duration of the P R interval is between 0 12 and 0 20 second

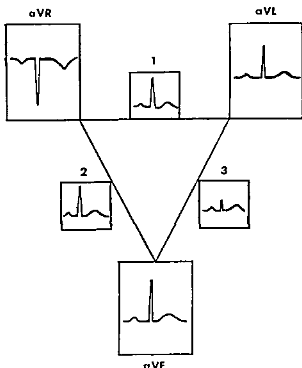


FIG 109 Scheme of the normal electrocardiogram In this as in similar sketches the amplitude of the waves in the standard limb leads has been made purposefully smaller in order to direct the attention toward the unipolar limb leads

The ventricular complex is composed of a rapid initial complex QRS and a slow final wave the T wave The beginning of the initial complex may be represented by a small Q wave This has been explained by the fact that depolarization starts on the left side of the septum and spreads from there toward the right side of this structure Therefore chest leads V5 and V6 present a small Q wave The small initial R wave of chest leads V1 and V2 has been explained as due to activation of either the septum or the right ventricle

After this the wave of depolarization spreads through the Purkinje fibers into the ventricular walls from the endocardium toward the epicardium This causes the upward deflection QR of the tracing As the left ventricular mass is greater its potentials are greater than those of the right the sum of depolarization of the ventricles is revealed as an upward deflection in V5 and V6 a downward deflection in V1 and V2

FIG 110 Duration and height of the different waves of the electrocardiogram Time relationship with the spreading of the stimulus through the various parts of the conduction system

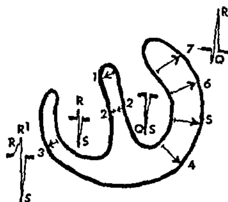


FIG 111 Order of activation of various fractions of myocardium (1 to 7) The arrows indicate the direction of spreading of the stimulus Intracardiac and epicardial ventricular complexes (inspired from an original of Wolff)

When depolarization has spread to the subepicardial layers the cavity and surface of the ventricles reach a stage of equilibrium and no changes of potential are recorded This phase of balancing of the forces is represented by the RS deflection For these reasons *QRS represents the period between beginning and completion of ventricular depolarization* (Figs 7 110)

The T wave reflects the stages of *ventricular repolarization* It is sometimes followed by a small positive wave the U wave, the mechanism of production of which is still obscure

The ecg has three isoelectric periods—periods which do not present any deflection (Fig 110) One is between the atrial complex (P) and the initial deflection of the ventricular complex (QRS) the second is between QRS and T the third is between T and the following P (the return to the base line may not be complete if diastole is short)

The atrial contraction takes place during the second half of P and during the first isoelectric period It can be measured approximately by the distance from the peak of P to that of Q

The ventricular contraction starts during QRS and lasts throughout the second isoelectric period and during T It can be measured approximately by the distance from the peak of Q to the end of T (so called Q T interval) According to an agreed standardization [1] the earliest upward wave is called R any downward preceding wave is called Q any downward following wave is called S If a second upward wave follows S it is called R′ and this may be followed by S (Fig 111 112)

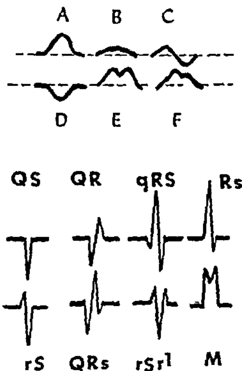


Fig 112 Variants of the P waves and of QRS complexes P waves *A* tall *B* flat *C* diphasic *D* inverted *E* bifid *F* notched

The Three Intervals of the Electrocardiogram

The P R Interval That part of the tracing which is between the beginning of P and the beginning of QRS is called the *P R interval* (it should be called more accurately *P Q*) The importance of this interval lies in the fact that it measures the time necessary for the atrioventricular conduction When the

P R interval is prolonged there is a disturbance of conduction either in the atrial myocardium or (more usually) in the tissues of the a v node and bundle of His This may occur (1) in congenital heart disease, (2) in acute myo carditis, (3) in myocardial fibrosis caused by coronary arteriosclerosis or following an acute inflammatory process, (4) after digitalis (5) during vagal stimulation A very common cause of prolongation of P R is rheumatic carditis *On the contrary P R is usually normal in the thyrotoxic heart and* in bacterial endocarditis

THE QRS INTERVAL This interval is measured from the beginning of the first rapid wave to the end of the last The QRS interval measures the time required by the stimulus to spread to and through the ventricles When pro longed it indicates impaired spreading of the stimulus either in the septum (bundle branch block) or within the ventricular wall (intraventricular block peri infarction block focal block) Exceptions are represented by ventricular premature beats, ventricular tachycardia and the pre excitation syndrome (Wolff Parkinson White syndrome)

THE Q T INTERVAL This interval measures the duration of electrical systole of the ventricles Its duration varies with the heart rate becoming shorter with a more rapid rate and vice versa Table 7 is constructed from figures of Hegglin and Holzmann [9]

TABLE 7 EXPECTED Q T TIME

Rate	*Average (Second)*	*Minimum*	*Maximum*
50	0 43	0 39	0 47
60	0 39	0 35	0 43
70	0 36	0 32	0 40
80	0 34	0 30	0 38
90	0 32	0 28	0 36
100	0 30	0 26	0 34
110	0 29	0 25	0 33
120	0 27	0 23	0 31

The figures of the expected Q T or average Q T were obtained by means of Bazett s formula $Q-T=K\sqrt{R\ R}$ * The actual Q T interval is measured in hundredths of second The ratio between the actual Q T and the expected $QT(r=\frac{\text{act } Q\ T}{\exp\ Q\ T})$ indicates whether or not the duration of electrical systole is prolonged Any ratio higher than 1 indicates prolongation Calculation of the ratio can also be made by the use of a nomogram [6]

ASPECT DURATION AND NOMENCLATURE OF THE DIFFERENT WAVES

P WAVE The P wave may be modified as far as voltage (tall or low) dura tion (broad) and configuration (notched slurred diphasic peaked or gothic)

* K is equivalent to 0 37 sec in men and 0 40 in women

(Fig 112) It is usually upright in Leads 1 and 2 may be occasionally inverted in 3 is frequently small or inverted in some of the chest leads and is normally inverted in aVR It is usually blunt and lasts from 0 06 to 0 11 second (Figs 110 112) in normal subjects

QRS COMPLEX The initial ventricular complex may be monophasic diphasic or polyphasic Slurring or notching is more common in aVF and in Lead 3 Normal amplitude is from 5 to 15 mm but it may be much larger in chest leads It lasts from 0 6 to 0 10 second in normal subjects The QRS complex has a different nomenclature according to the direction of the first wave and to the number of subsequent waves This can be conveyed to the reader by using capitals or small letters according to the height of the waves and by giving to each wave its symbol in sequence (Fig 112)

T WAVE The T wave may be modified as far as voltage (tall or low) polarity (positive or negative), and configuration (diphasic symmetrical or asymmetrical) (Fig 113) It is normally upright However it is normally inverted in aVR and may be inverted in aVL it is normally inverted in V1 and V2 (or even V3 and V4) in infants It lasts from 0 16 to 0 25 second (Figs 110 113)

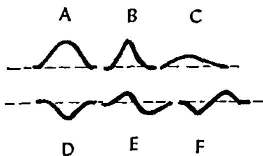


FIG 113 Various types of T waves *A* tall *B* peaked *C* low *D* inverted *E* diphasic (+ −) *F* diphasic (− +)

S T The S T segment may be isoelectric (normal) or displaced upwards or downwards The direction and form of the S T segment may indicate the difference between the coronary (or cove shaped) T wave and the digitalis T wave (Fig 115) It normally follows the baseline but may be slightly raised (not more than 1 mm =0 1 mv) The displacement may reach about 2 mm in the chest leads (Fig 115)

SPECIAL FEATURES OF CERTAIN LEADS

Unipolar Right Arm Lead (VR aVR or R)

The ecg presents typically inverted waves the P wave the main rapid ventricular deflection and the T wave are directed downwards (Fig 109) The heart has its base directed upwards and toward the right the thin vascular and atrial walls contribute little to the tracing and leave free passage to the stronger ventricular potentials The stimulus after reaching the septum and the endocardial surface of the ventricular walls spreads outwards toward the epicardium This causes a vector directed away from the electrode (so called *negative potentials within the cavities*) and explains the inverted QRS

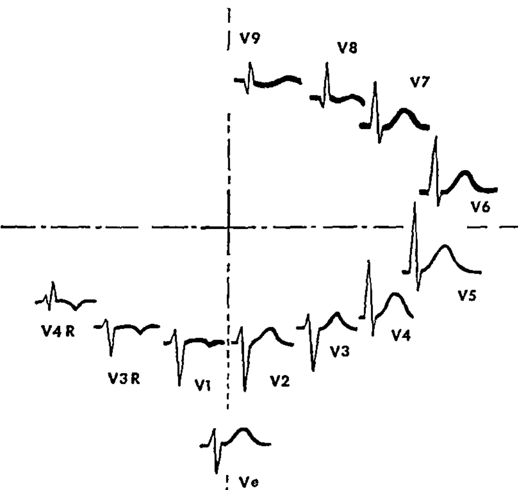


FIG 114 The electrocardiogram in the chest leads V1 to V9 and in VE Normal subject with vertical heart

and T waves of VR (Fig 111) The QRS complex is similar to that obtained by introducing an electrode within the upper part of the right atrial cavity The currents of both ventricles are recorded This lead is only slightly influenced by the position of the heart within the chest

Unipolar Left Arm Lead (VL aVL or L)

This lead usually has positive waves The picture varies considerably, depending on changes in position of the heart with subsequent shifting of the electric axis (p 257) It reflects the variations of electric potential presented by the anterior and lateral epicardial surfaces of the heart

Unipolar Left Leg Lead (VF aVF or F)

This lead usually has positive waves It is strongly influenced by the position of the heart within the chest and by the resulting shifting of the electric axis

of the heart (p 257) It reflects the variations of electric potential presented by the posterior and diaphragmatic epicardial surfaces of the heart

Standard Leads (1 2 and 3)

These present normally positive waves P may be diphasic in 3 QRS may have a deep S in 1 or an rS in 3 if the electric axis of the heart is shifted to an unusual degree (p 257) T may be inverted in 3 due to the same cause (Fig 114)

Chest Leads

The anterior surface of the heart including parts of both ventricles lies closer to the surface of the body (anterior chest wall and left axilla) than any other part of the heart Leads from various anterolateral points of the chest (V1 to V6) resemble direct epicardial leads more than any other lead from the body surface (Figs 111 and 114) Still it should be kept in mind that they are not identical because the tracing is not completely dominated by a small fraction of muscle lying in proximity to the electrode and includes potentials from the surrounding areas For this reason an *intrinsic deflection** cannot be recognized in a chest electrocardiogram

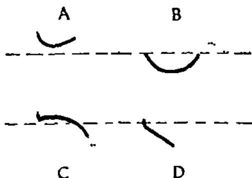


Fig 115 Variations of the S T segment *A* displaced upwards *B* displaced downwards *C* cove shaping of S T typical of coronary lesion *D* typical of digitalis effect

V1 and V2 give the best information about right ventricular potentials VE records the potentials of the diaphragmatic or posteroinferior aspect of both ventricles (Fig 114) The R wave is smallest in V1 and increases gradually in height from V1 to V5 or V6 where it is tallest The S wave is deepest in V1 or V2 and decreases gradually from right to left until it disappears Q waves are present over the left side of the precordium but are small Normal QRS should not last more than 0 10 second In V1 the intrinsicoid deflection begins 0 006–0 02 second after the onset of QRS in V5 or V6 it starts about 0 035 second after it The T waves are upright in all leads but may be inverted in V1 (occasionally in V2) It should be noted that in certain normal subjects V1 and V2 seem to present an inverted initial deflection (type

* *Intrinsic deflection* is the name given to the sharpest and largest downward deflection which represents depolarization of the muscle under an electrode The similar deflection recorded by the chest leads is called *intrinsicoid deflection* and it is supposed to represent depolarization of the heart muscle in the area which is *nearest* to the electrode

TABLE 8 CHARACTERISTICS OF ELECTROGRAPH LEADS

Type and Symbol of lead	Origin of currents recorded
Unipolar limb leads	
VR	Endocardial surface (both ventricles)
VL	Epicardial surface of anterior and lateral walls (mainly left ventricle)
VF	Epicardial surface of postero diaphragmatic wall (both ventricles)
Bipolar (standard) limb leads	
1	Endocardial epicardial lead (VL VR)
2	Epicardial endocardial lead (VF VR)
3	Epicardial epicardial lead (VF VL)
Standard unipolar limb leads	
V1	Epicardial surface of right atrium plus epicardial surface of right ventricle
V2 V3	Anterior epicardial surface of right ventricle
V4	Transitional zone (occasionally displaced)
V5	Low anterolateral epicardial surface of left ven tricle
V6	Low lateral and posterolateral epicardial surface of left ventricle
High unipolar chest leads	
High V1	Epicardial surface of right atrium Endocardial surface of ventricles
High V5	High anterolateral epicardial surface of left ventricle
High V6	High lateral and posterolateral epicardial surface of left ventricle
Posterior unipolar chest leads	
V7 V8 V9	High posterior epicardial surface (left ventri cle)
Ensiform unipolar lead	
VE	Diaphragmatic epicardial surface (both ventri cles)
Esophageal unipolar leads*	
VEs 35 40	Left atrial epicardial surface left ventricular endocardial surface
VEs 45 55	Posterobasal epicardial surface (both ventricles)

* Details of the esophageal leads will be given in Chapter 30

QS) on account of the extreme smallness of R The S T segment may be slightly above the baseline this displacement usually does not exceed 1 mm but may reach 2 mm (Fig 115)

Table 8 provides a key to the interpretation of the various leads

NORMAL AND ABNORMAL ELECTROCARDIOGRAMS

It is impossible to draw a sharp line between normal and abnormal tracings In reaching a decision the observer must consider the possible occurrence of a certain variation in the ecg of a normal person Statistical tables have been made and reference to them may be useful However personal experience and additional clinical and laboratory data determine whether or not an ab normality should be considered pathologic

GENERAL RULES OF REGISTRATION

Application of electrocardiography to large numbers of hospital patients has resulted in the fact that recording developing and mounting of the trac ings is usually done by technicians This practice justified by the need for mass work has been followed also in private laboratories and even in the doctor s office

An *electrocardiographic report* can be of different types (1) purely de scriptive (2) including analysis of certain peculiarities like axis deviation premature beats etc (3) including a diagnosis such as atrial flutter *myocardial infarction digitalis heart*

A complete electrocardiographic evaluation can be done only by a cardi ologist who has examined the patient Vague generalities like coronary heart disease or rheumatic heart do not add much to existing knowledge and moreover are often not accurate The study of multiple precordial leads emphasizes the need of a close supervision of the technician

PRACTICAL APPLICATIONS

1 Electrocardiography may be useful for an exact evaluation of the atrial and ventricular rates and for the location of the pacemaker Thus diagnosis of abnormal rhythms and their focus of origin is made possible

2 The electric axis can be determined dextrocardia can be diagnosed The data are of help in the study of congenital malformations valvular disease chronic cor pulmonale etc

3 Delayed a v conduction a v block bundle branch block or intraventric ular block are recognized

4 Evidence of diffuse myocardial damage is obtained This includes infectious lesions and digitalis effect

5 Evidence of localized myocardial ischemia or damage is secured This is of great importance for the diagnosis of coronary heart disease and the differential diagnosis with pulmonary infarction, dissecting aneurysm of the aorta and pericarditis

It shall be emphasized that, in most conditions, repeated registration of electrocardiograms is necessary, interpretation will be far more accurate than after a single tracing

In requesting an electrocardiogram the physician should give the following information (1) presence of valvular defects, (2) level of blood pressure (3) administration of digitalis or allied drugs or other drugs acting on the myocardium (quinidine emetine insulin), clinical diagnosis or reason for asking a tracing

The time relationships between the electrocardiographic waves and those of other tracings are as follows

1 Initial evidence of right atrial contraction (mechanical cardiogram electrokymogram atrial sound by esophageal route) is found 0 04–0 06 after the rise of P The atrial sound recorded at the apex follows the peak of P by a longer interval

2 The beginning of any evidence of ventricular contraction (mechanical cardiogram electrokymogram first sound at apex) is found between Q and R coinciding with R or between R and S

3 The end of the T wave usually coincides with the end of mechanical systole of the ventricles However a dissociation between the two phenomena may occur in abnormal conditions

Name______________________________Age________

Clinic_______________Clinic #____________Date__________

Referring Physician___________________________________

Tentative Diagnosis___________________________________

Blood pressure_______________Digitalis yes no

Previous ecg date_______________No____________________

Appointment________(Day)________(Date)______________(Time)

Fig 116 Request for electrocardiogram

Name________________ Age______ Date__________

Clinic________________ Clinic #__________

Rhythm________________ Rate__________

P________________ P R__________

QRS__________________________

ST__________________________

T__________________________

Axis Shift________________ Ventricular hypertrophy__________

Ischemia__________

Ecg Interpretation__________________________

____

Clinical Correlation__________________________

____

Signed__________

Fig 117 Report of electrocardiogram

4 There is no relationship between the U wave and the phase of rapid ventricular filling

Suggested forms for a request for an electrocardiogram and electrocardio graphic report are reproduced in Figs 116 and 117 Both are in use in the author s laboratory

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CHAPTER 30

Special Electrocardiographic Technics

ESOPHAGEAL ELECTROCARDIOGRAM

History

Waller[3] recorded the first esophageal electrocardiograms in man in 1889 followed by Cremer[3] in 1906 Lieberson and Lieberson[11] in 1934 used a bipolar lead (esophagus-left leg) for clinical studies A local bipolar lead (esophagus esophagus) was used by the author in 1935[14] for the study of left atrial potentials Further studies with bipolar leads were made by Brown (esophagus-left leg) and by Ramirez and co workers[13] (precordium esophagus) Nyboer[18] in 1941 gave a complete description of the tracings at various levels using unipolar esophageal leads Scherlis and co workers[19] further studied the ventricular electrocardiogram by the esophageal route

Technic

A thin rubber tube ending with a metal tip and containing the connecting wire is introduced through the nose or mouth (preferably under fluoroscopic control) The tip is used as an exploring electrode while the indifferent electrode is represented by Wilson s central terminal Thus the tracings are unipolar esophageal leads and should be marked with the symbols VEs and a notation of the depth of the electrode from the nostrils or the dental arch

A different method employed by the author[14] made use of a rubber tube with two silver rings at 10 mm from each other and two internal wires This

bipolar esophageal lead presents certain advantages because it gives larger atrial deflections

The technic for the introduction of the esophageal electrode is the same as that for the tube in a stomach wash The patient is seated with the head slightly tilted forward The tube can be introduced either through one of the nostrils or through the mouth In case of violent gag reflexes, the throat can be swabbed with a 2 per cent cocaine hydrochloride solution The tip of the tube is passed through the pharynx while the patient swallows a small amount of water After introduction the patient may be placed in the recumbent position and he is invited to breathe deeply if gagging is still present An electrical filter has been used in order to exclude slow currents due to respiration and esophageal peristalsis In general, however it can be dispensed with

The following four standard esophageal leads should be studied[3] (Fig 118) (1) VEs 30–35 atrial lead, (2) VEs 35–40 low atrial lead, (3) VEs 40–45 ventricular lead, (4) VEs 45–55, low ventricular lead

FIG 118 Unipolar electrocardiograms from the esophagus *A*, at E 35 *B*, at E 39 *C* at E 40 *D* at E 45

Analysis of Waves

At the atrial level (Postion 1), the P wave is prominent and shows an intrinsic deflection similar to that usually seen in the ventricular complex[11] A deep inverted initial ventricular deflection and an inverted T wave are normally seen The nearness with the left atrium explains the high voltage and the diphasic aspect of the atrial complex The ventricular waves are inverted and indicate cavity potentials which spread through the thin atrial wall In Position 2 transitional tracings are obtained

In Position 3, a different pattern is usually observed It consists of a positive P an initial ventricular complex with a qR or QR pattern and a diphasic or positive T wave Considerable differences are noted according to the position of the heart (p 271)

In Position 4, the electrode has reached the diaphragmatic surface of the heart and records a tracing similar to VE or aVF a positive P variable QRS patterns usually positive but occasionally inverted T waves

The differentiation between a Q caused by normal cavity potentials and a Q due to posterior myocardial infarct is based on the fact that, in the first

instance P is positive has a high voltage and an 'intrinsic deflection T is inverted In the case of the infarction there may be a QS or a QR pattern, and T may be upright or inverted However this differentiation is not always easy

Conclusions

Esophageal leads are useful for the study of atrial and interatrial disorders (Position 1) and for the recognition of coronary disturbances especially myo cardial infarction involving the posterodiaphragmatic aspect of the heart (Position 3)

INTRACARDIAC ELECTROCARDIOGRAM

History

Intracardiac electrocardiograms in open chest experiments were recorded by several authors following the studies of Lewis[10] The first intracardiac tracings by means of catheterization of the right heart were recorded in the dog by the author and his co workers[15] in 1937 Clinical studies by means of right heart catheterization were made by Lenegre and Maurice[8] in 1945 Hecht[6] in 1946 Battro and Bidoggia[1] Sodi Pallares *et al*[21] in 1947 and Duchosal *et al*[4] in 1948 The first clinical study of ventricular potentials by catheterization of the left heart was done by Sodi Pallares *et al*[20] in 1950

Technic

A catheter for cardiac catheterization with a small silver electrode con nected to the silver mandril is introduced into the cavities of the right heart following the usual technic of right heart catheterization (p 163) The posi tion of the tip of the catheter is determined by means of fluoroscopy pressure readings and oximetry The endocardial electrode is connected with the wire commonly used for chest leads and serves as an exploring electrode The central terminal completes the circuit

Analysis of Waves

Intracardiac (endocardiac) leads can be considered as direct leads from the endocardial surface of the heart The *atrial electrogram* consists of a series of rapid deflections (S e i o waves) and a final slow wave (Ta) (Fig 119) S has been considered as due to the activity of the s a node and precedes by 0 05 second the onset of P in the limb leads The Ta wave represents the final phase of repolarization of the atria and is visible only in cases of a v block The interval from the beginning of P in the limb leads to the end of Ta measures from 0 34–0 42 second but may be much longer

The *right ventricular electrogram* may be recorded directly from the ventricle (Fig 120) it shows an initial positive deflection (R) due to

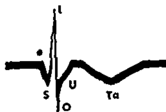


Fig 119A Intracardiac electrogram from the right atrium in man (there is no ventricular complex on account of a v block) (From originals of Battro and Bidoggia)

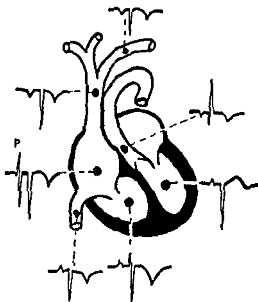


Fig 119B Intracardiac electrograms in a normal man

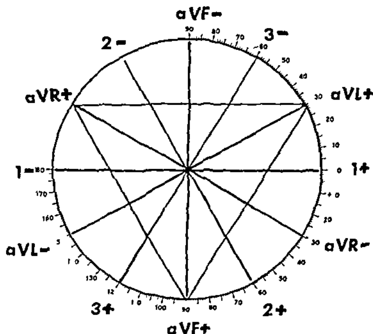


Fig 120 Determination of the electrical axis by the system of the six axes

depolarization of the septum (and possibly of the right ventricle) a large inverted deflection (S) and an inverted T wave If the ventricular electrogram is recorded from the atrium a QR or QS complex and a deep pointed inverted T wave are present

The *left ventricular electrogram* (Fig 119B) shows a QS type of initial ventricular complex there may be some degree of elevation of S T possibly due to pressure of the catheter The P wave is positive Above the aortic valve the electrogram has an atrial type and presents negative P and T waves and an initial complex of the qR type

Conclusions

Intracardiac electrocardiography has an important place in research but limited clinical applications Rare electrocardiographic anomalies may require an intracardiac tracing for explanation

INTRABRONCHIAL ELECTROCARDIOGRAM

History

Bipolar intrabronchial electrocardiograms were recorded by Savjaloff[7] in a laryngectomized patient Langner and Atkins[7] and Goldstein and co workers[5] have studied intrabronchial electrocardiograms with unipolar leads

Technic

Following mild sedation with barbiturates and local laryngeal anesthesia a bronchial electrode is introduced by a laryngologist The catheter is introduced first into an anterior branch of the right lower bronchus then into several other bronchi of the right side then into several bronchi of the left side The central terminal completes the electric circuit

Analysis of Waves

The electrograms recorded within the right bronchi are similar to those recorded within the right atrium Those recorded within the left bronchi vary according to the position they may reveal cavity potentials (upper or medium left) or a qR type of ventricular complex with an upright T (low anterior bronchus) similar to the patterns of V6 V7 or V8

Conclusions

This method is useful only in special cases or for research It affords wide exploration of the left ventricular epicardial surface and provides information similar to but more complete than that obtained by the esophageal route

CONTINUOUS ELECTROCARDIOGRAM

Clinical cases may present occasional or sporadic paroxysmal phenomena which are of difficult study only because they are not present at the time of an electrocardiographic examination These phenomena frequently occur at night and may have a brief duration The study of these cases has been made possible by an apparatus which records continuously the electric currents of the heart on a microfilm (Likoff *et al*[6]) The study is prolonged for twenty four hours and during the patient s sleep without any assistance Subsequently, the film is examined by means of a magnifying device the most interesting sections are selected and photographically magnified for study

FUNCTIONAL TESTS

Several functional tests have been suggested in order to evaluate the efficiency of the coronary system They can be used whenever the history of a patient seems to indicate coronary heart disease while the electrocardiogram is normal The most used are the anoxemia test and the exercise test

Anoxemia Test

It has been advocated by R Levy and co workers[9] since 1941 and is per formed in the following way The patient inhales a mixture of 10 per cent oxygen with nitrogen which is insufficient for the normal oxygen supply to the tissues A tracing is recorded before the test and at five minute intervals dur ing the inhalation for twenty minutes Normal persons have only slight electro cardiographic changes consisting of decreased height of T in all leads and occasionally an inversion of T in Leads 2 and 3

A positive test consists of the following changes

1 The sum of S T deviations in leads 1, 2 3, and V4 should be greater by 3 mm or more than in the control tracing
2 There is a partial or complete reversal of the direction of the T wave in 1 accompanied by S T deviation of 1 mm or more in this lead
3 *There is a complete reversal of the direction of the T wave in V4 regardless* of any associated S T deviation in this lead

A tank of 100 per cent oxygen is incorporated in the circuit so that oxygen can be rapidly administered if needed

The "Two Step Exercise Test

This test has been described by Master and Oppenheimer[16 13] in 1929 Since normal persons submitted to excessive exercise may present electro cardiographic changes the exercise has been standardized for age weight and sex The procedure is the following The weight of the subject and a con

TABLE 9 STANDARD NUMBER OF ASCENTS FOR MALES FOR MASTER'S TEST

Weight (lb)	*Age in Years*												
	5–9	*10–14*	*15–19*	*20–24*	*25–29*	*30–34*	*35–39*	*40–44*	*45–49*	*50–54*	*55–59*	*60–64*	*65–69*
40–49	35	36											
50–59	33	35	32										
60–69	31	33	31										
70–79	28	32	30										
80–89	26	30	29	29	29	28	27	27	26	25	25	24	23
90–99	24	29	28	28	28	27	27	26	25	25	24	23	22
100–109	22	27	27	28	28	27	26	25	25	24	23	22	22
110–119	20	26	26	27	27	26	25	25	24	23	23	22	21
120–129	18	24	25	26	27	26	25	24	23	23	22	21	20
130–139	16	23	24	25	26	25	24	23	23	22	21	20	20
140–149		21	23	24	25	24	24	23	22	21	20	20	19
150–159		20	22	24	25	24	23	22	21	20	20	19	18
160–169		18	21	23	24	23	22	22	21	20	19	18	18
170–179			20	22	23	23	22	21	20	19	18	18	17
180–189			19	21	23	22	21	20	19	19	18	17	16
190–199			18	20	22	21	21	20	19	18	17	16	15
200–209				19	21	21	20	19	18	17	16	16	15
210–219				18	21	20	19	18	17	17	16	16	14
220–229				17	20	20	19	18	17	16	15	14	13

* An ascent is one complete trip over the steps in one direction.

TABLE 10 STANDARD NUMBER OF ASCENTS FOR FEMALES FOR MASTER'S TEST

Weight (lb)	*Age in years*												
	5–9	*10–14*	*15–19*	*20–24*	*25–29*	*30–34*	*35–39*	*40–44*	*45–49*	*50–54*	*55–59*	*60–64*	*65–69*
40–49	35	35	33										
50–59	33	33	32										
60–69	31	32	30										
70–79	28	30	29										
80–89	26	28	28	28	28	27	26	24	23	22	21	21	20
90–99	24	27	26	27	26	25	24	23	22	22	21	20	19
100–109	22	25	25	26	26	25	24	23	22	21	20	19	18
110–119	20	23	23	25	25	24	23	22	21	20	19	18	18
120–129	18	22	22	24	24	23	22	21	20	19	19	19	17
130–139	16	20	20	23	23	22	21	20	19	19	18	17	16
140–149		18	19	22	22	21	20	19	19	18	17	16	16
150–159		17	17	21	20	20	19	19	18	17	16	16	15
160–169		15	16	20	19	19	18	18	17	16	16	15	14
170–179		13	14	19	18	18	17	17	16	16	15	14	13
180–189			13	18	17	17	17	16	16	15	14	14	13
190–199			12	17	16	16	16	15	15	14	13	13	12
200–209				16	15	15	15	14	14	13	13	12	11
210–219				15	15	14	14	13	13	13	12	11	11
220–229				14	13	13	13	13	12	12	11	11	10

trol electrocardiogram are recorded Then the patient is invited to walk up one side of a two step stool* and down the other He makes a trip only when a count is given and the required number is completed in one and one half minutes The electrocardiogram is then recorded at two and six minutes after cessation of the exercise The standard numbers of ascents for males and females are presented in Tables 9 and 10

Criteria for an abnormal electrocardiographic response are

1 Depression of S T of more than 0 5 mm below the isoelectric level in 1, 2 or V4
2 Change from an upright T wave to an isoelectric or inverted T wave (or becoming upright of a previously inverted T)
3 Onset of arrhythmias immediately after exercise

Amyl Nitrite Test

This test has been described by Contro Haring and Goldstein [4] in the author's laboratory After a short rest in recumbent position the electrocardiogram is recorded Then an ampule of amyl nitrite is broken and placed under the patient s nostrils and the patient is instructed to breathe deeply As soon as flushing of the face occurs an electrocardiogram is again recorded and the tracing is repeated at 1 minute intervals or less until it becomes normal

The sudden drop of blood pressure caused by amyl nitrite determines coronary insufficiency whenever the coronary arteries are sclerotic Evidence of abnormal response is displacement of S T of more than 1 mm from the baseline flattening or inversion of the T wave in any lead and premature contractions This test is harmless because the ischemia is of short duration and the vasodilating properties of the drug prevent functional disturbances The result of the test usually coincides with that of the two step exercise test

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* This stool is made of two steps having a height of 22 cm (or 9 inches) It is easily built by a carpenter

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CHAPTER 31

The Electrical Axis, Vectorcardiogram, Ventricular Gradient

ELECTRICAL AXIS

According to an assumption of Einthoven [6] the right shoulder left shoulder and left hip form the apices of a triangle If it is also assumed that they are equidistant from the center of the triangle and that the heart is located at this center if it is further admitted that the conducting media are homogeneous various important calculations can be made from the tracings recorded in the standard limb leads

Positive and negative charges (dipoles) appear on the surface of a cell whenever there is a change in the permeability of its membrane Each charge can be thought of as exerting a force and its magnitude is proportional to the strength of the charge Each charge exerts its greatest force along a line which connects the charges (from the negative to the positive) and which represents the direction of the force These two elements magnitude and direction as well as sense have been used in order to represent the charges as *vectors*

The electrical field around the heart is the result of first depolarization and then repolarization of several fibers the combined effects can be found by determining the sum of the vectors representing the various charges or *manifest vector* The force resulting from this vector can be easily determined by drawing perpendicular lines from the origin and end of the vector line to

each side of the triangle These perpendiculars indicate the magnitude and sense of the deflections in leads 1, 2, 3 at any instant At the beginning of depolarization, the septum is activated from left to right (Figs 7, 111), at the end of this phase the posterobasal region of the left ventricle is activated from right to left This causes the various vectors forming the initial QRS complex The phenomena associated with repolarization and causing the T wave can also be projected onto the three lines resulting in another vector

The concept of the equilateral triangle (a rough approximation) was used by Einthoven *et al*[5] in order to calculate the magnitude of electromotive forces of the heart as projected on the frontal plane of the body Determination of the *electrical axis* and measure of the axis deviation on the basis of the above method have been made in clinical cases for several decades The procedure is the following

1 The magnitudes of either P, QRS or T in leads 1 and 3 (in mv) are traced upon two sides of an equilateral triangle inscribed within a circle marked in degrees in order to determine the mean axes of P QRS or T That of QRS is the most commonly studied (Fig 122)

2 Perpendicular lines are drawn from the points obtained

3 A line is drawn from the center of the triangle to the point of intersection of the perpendiculars This last line represents the direction and magnitude of the electrical forces projected upon the frontal plane of the body

4 The line drawn in (3) is prolonged to intersect the circle and the degree of inclination of the axis is read directly on the circle

In general the term electric axis' is used as applied to the axis of QRS Degrees from 0 to +90 represent normal axis degrees from 0 to −90 represent left axis deviation, degrees from +90 to +180 represent right axis deviation A right axis deviation indicates that the potentials of the left arm are lower than those of the right A left axis deviation indicates that they are higher

It should be pointed out that determination of the axis by plotting the *direction and height of the waves is a practical but only approximate procedure* A correct procedure should be based on plotting both the direction and the surface of the waves

Calculation of the electrical axis can be based also on the waves of the unipolar limb leads Modifying a scheme of Bayley[3] (so called *system of the three axes*) Sodi Pallares *et al*[11] have described a *system of six axes* which permits determination of the electrical axis by the use of either the standard or the unipolar limb leads In this system three lines or axes represent the values of the standard leads (1 2 3) and three other lines represent those of the unipolar limb leads (R L F) (Fig 120) The values of the waves (in square mm) are placed along the lines, perpendiculars are drawn, then a line

is drawn from the center to the point of intersection of the perpendiculars and prolonged until it intersects the circle

Deviations or shifts of the electric axis are not now considered as important as they once were

VECTORCARDIOGRAM

As we have said the line indicating the axis has direction magnitude and sense Therefore it can be called a *vector* Such a vector can be constructed for each of the points and for each of the waves of an electrocardiogram All vectors have their origin at zero and vary in direction and size according to the changes of the electrical field of the heart recorded in the electrocardiogram Each of these vectors is called an *instantaneous electrical axis* The end points of the various vectors can be connected by a line which describes a loop for each electrocardiographic wave (loop of P loop of QRS loop of T) (Fig 121) The largest diameter of each loop is identical with the *modal electrical axis* of a given deflection

The loop connecting the end points of all possible instantaneous axes called *frontal vectorcardiogram* has an irregular shape and can be constructed graphically (Fig 122) However a simpler way is that of connecting the electrocardiographic wires with an oscilloscope Then the light beam of a cathode ray tube traces automatically the outline of the loop on the fluorescent screen A photograph of this loop may be used to reproduce the vectorcardiogram

Spatial Vectorcardiography

The first concept of spatial vectorcardiography was introduced by Mann[8] in 1920 The same author successfully recorded the vectorcardiogram by means of a special galvanometer[9] Sulzer and Duchosal[1] Schellong[10] and Wilson and Johnston[13] recorded the vectorcardiogram by means of a cathode ray oscilloscope Various geometric arrangements have been suggested for placing the electrodes necessary to record the various vectors not only in the frontal place but also in the anteroposterior plane The best known are

1 The method of the equilateral tetrahedron suggested by Wilson Johnston and Kossmann[14]
2 The method of the double cube suggested by Duchosal and Sulzer[4]
3 The method of the sagittal triangle suggested by Arrighi[1]
4 The method of the cube advocated by Grishman Borun and Jaffe[7]

The method of the cube is based on the following technic It is assumed that the central origin of all electrical forces created by the heart is at the center of a sagittal plane passing just to the left of the sternum at the level of

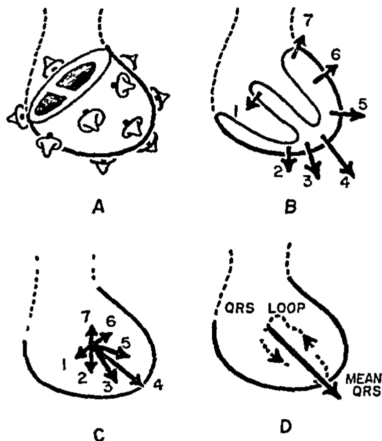


Fig 121 The QRS vectors of the heart *A*, Each QRS vector is directed perpendicularly to the surface of the region where it is generated The T vectors have a similar direction in the normal subject *B* Frontal plane cross section of the heart illustrating the magnitude, direction and effective source of resultant QRS vectors from instant to instant during a single QRS cycle *C*, The instantaneous vectors from the previous figure are drawn as if they all originated at the same point the relative zero point of the electrical field in the frontal plane *D* The pathway of the terminus of the QRS vector from instant to instant is a single QRS cycle (QRS loop) and the mean of the instantaneous vectors (mean QRS vector) is projected in the frontal plane of the body (From Grant and Estes *Spatial Vector Electrocardiography* courtesy of the Blakiston Co)

the fourth intercostal space Four electrodes are placed at four corners of a cube having this point at its center

1 One electrode (with three cables) is placed near the right posterior axillary lines at the level of the first and second lumbar vertebrae
2 One electrode (with one cable) is placed near the left axillary line at the same level
3 One electrode (with one cable) is placed vertically over the right scapula
4 One electrode (with one cable) is placed on the right anterior axillary line at the level of the first and second lumbar vertebra

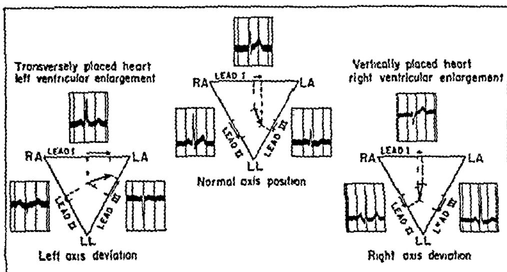


FIG 122 Frontal plane vectorcardiograms in *Left* a case of hypertensive heart disease with left axis deviation *center* a normal subject *right* a patient with mitral stenosis and right axis deviation (From H Hecht *Basic Principles of Electrocardiography* courtesy of Charles C Thomas publisher)

Thus there are three electrodes at the level of the first second lumbar vertebrae (two posterior one anterior and to the right) three posterior electrodes (two at the level of the first second lumbar vertebrae one over the right scapula) and one anterior electrode (on the anterior axillary line at the level of the first second lumbar vertebrae)

Three bipolar leads are used

1 One recording the horizontal component (negative pole at the posterior axillary line positive pole at the left posterior axillary line)
2 One recording the vertical component (negative pole at the right shoulder positive pole at the right posterior axillary line)

3 One recording the sagittal component (negative pole at the right posterior axillary line, positive pole at the right anterior axillary line)

The three amplifiers of a three channel electrocardiograph are arranged to feed directly the plates of a triple cathode ray oscilloscope The same degree of amplification is used for all three channels and the record is taken with the patient in the horizontal position Introduction in the circuit of 1 mv causes the beam to deviate 45 degrees downward and to the right of the ob server

The loops occurring in the three planes are then recorded simultaneously by a camera Therefore, three vectorcardiograms are recorded at the same time for each subject (Fig 123)

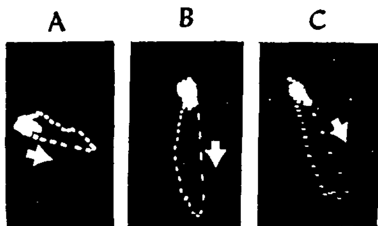


Fig 123 Spatial vectorcardiogram *A* frontal plane *B*, sagittal plane, *C*, horizontal plane (From Grishman Borun and Jaffe, courtesy of the C V Mosby Co)

The vector method represents a different point of view in comparison with other methods of interpretation of the electrocardiogram even when the same *electrode positions are used and the same deflections are studied* The vector method has the following advantages over empirical methods basing their interpretation on the pattern of the various leads [6]

1 It simplifies clinical interpretation by eliminating the need to memorize patterns
2 It is more accurate and objective because the deflections are reduced to a few simple measurements of the electrical field
3 It is easier to separate normal tracings and positional changes from trac ings caused by myocardial abnormalities

However, vectorcardiography can only supplement and should not sup plant, conventional methods of pattern interpretation

VENTRICULAR GRADIENT

The studies of Wilson and co-workers[15] and those of Ashman and co workers[2] have established the concept of "ventricular gradient" and its clinical appreciation

In general the term *gradient* (G) is used to indicate the rapidity of a variation (like those of pressure temperature or electricity) As this variation is characterized by three values e g sense direction and magnitude it complies with the definition of a vector

When a muscular fiber is stimulated the currents generated by the stimulus can be theoretically considered as the resultant of two monophasic waves of opposite polarity Comparison of the surfaces or areas (A) of these waves shows that they are identical Being of opposite polarity their algebraic sum is zero and there is no gradient If the waves have a different duration then the algebraic sum of the areas of the two waves is not equal to zero and represents a gradient of value x

In the mammalian heart several conditions (high pressure low blood supply) contribute to slow down the process of depolarization in the sub endocardial layers Therefore the subendocardial layers which were depolarized first are repolarized last For this reason the areas of QRS (A QRS) and T (AT) which represent the resultant of the various vectors of depolarization and repolarization in the subendocardial and subepicardial layers have surfaces of different values The difference between the two surfaces is the ventricular gradient A similar comparison can be repeated for the atrial potentials in order to study whether or not there is an atrial gradient

A change of the area of T (AT) may be the result of changes of the area of QRS (A QRS) because variation of the order of depolarization of the various bundles is necessarily followed by a corresponding change in their order of repolarization In other words a change of the area of T may be the result of an abnormality of QRS as in ventricular hypertrophy bundle branch block ventricular extrasystoles or the preexcitation syndrome there is a *secondary change of T* and the gradient is normal On the contrary a change of the area of T may be due to abnormal metabolism of certain areas of the myocardium which does not affect QRS there is a *primary change of T* and this is revealed by a modification of the ventricular gradient

A wave or deflection of the electrocardiogram may be defined by its duration in seconds and by its height in microvolts (or tenths of a millivolt) These two factors determine the area included in one wave The same geometrical construction which is used for determination of the electrical axis may be employed in order to obtain the area of a certain wave from the areas found in two standard limb leads The area of QRS (called A QRS) is the mean electrical axis of QRS is two-dimensional and is identical with the

frontal vectorcardiogram for QRS The area of T (A T) is the mean electrical axis of T and has the same respective properties

The ventricular gradient is measured in the following way (1) The areas of QRS and T are measured with the help of a magnifying lens * (2) The vectors of these areas are plotted on the lines of a triangular system, and perpendicular lines are drawn (3) The line drawn from the center of the triangle to the point of intersection of the perpendiculars gives the degree of inclination of the gradient

In the normal heart the mean value of the ventricular gradient is 13 AU the minimum value is 2 5 AU, and the maximum value is probably above 23 AU The gradient forms an angle between −53 and +136 7 Approximations of ±15 per cent for the values and ±5 per cent for the angle should be taken into consideration

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* Each small square represents a time voltage unit (0 1 mv × 0 04 second) called the *Ashman Unit* (AU) One fourth of this is a microvolt second resulting from a height of 100 mv by a time of 0 01 second Since the ecg waves (QRS and ST T) are grossly triangular their widths and heights are multiplied and the result *is divided by two in order to obtain* the area in Ashman Units

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CHAPTER 32

The Electroangiogram

HISTORY

Electrical phenomena of the arterial wall were recorded by Tigerstedt,[10] Huerthle[4] and Bittorf[1] in experimental animals They were considered as action currents of the smooth muscular fibers of the arteries Blumenfeldt[2] obtained electric currents from dead perfused arteries and discounted previous studies The author[7] proved later that killed arteries present currents only when perfused through inadequate systems, creating artifacts not when the fluid and the arterial wall have the same electrolyte content and the perfusion system does not originate currents Surviving perfused arteries present slow deflections reproducing the form of the pulse and may present rapid deflections if stimulated by drugs[8] Slow deflections from the femoral artery of animals detached from the body but perfused by the same animal were further recorded[9] These electrical phenomena of the arterial wall have been recorded also by Huerthle[5] on the aorta and by von Dungern[3] following electrical stimulation of peripheral arteries Electroangiograms from the radial artery of man were recorded by the author in 1931[6]

TECHNIC

As the magnitude of the arterial potentials is between 0 05 and 0 2 mv registration can be made only by an electrocardiograph connected with a pre

amplifier which is capable of magnifying the currents from ten to twenty times Theoretically one could use the central terminal as one electrode However a bipolar lead is preferable because it avoids AC interference The electrodes are represented by silver coated needles Following local antisepsis these needles are introduced through the skin to the proximity of either the radial or the femoral artery Care should be taken not to injure the vessel

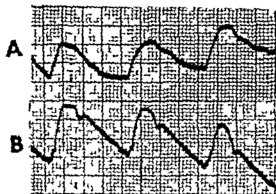


FIG 124 *A* Femoral electroangiogram in man (right side) *B* femoral pulse (left side)

ANALYSIS OF WAVES

The tracing obtained in man resembles that of a pulse tracing over the same artery (Fig 124) As the described technic is based on the introduction of a foreign material near the artery it is probable that the tracing is due to a combination of action currents (which develop during the tonic dilatation and contraction of the artery) and physicochemical currents

CONCLUSIONS

The method can be used for research but needs further investigation

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PART III

Physiologic Variants of the Graphic Tracings

CHAPTER 33

Tracings in Normal Adults

THE ELECTROCARDIOGRAM IN THE VARIOUS POSITIONS OF THE HEART

Electrocardiograms of normal subjects were recorded for many years by means of the standard limb leads It soon became apparent that the patterns of certain normal subjects were similar to those of patients with abnormal hearts In particular the ecg of the vertical heart sometimes was confused with that of right ventricular hypertrophy that of the horizontal heart with the tracing of left ventricular hypertrophy A new era was opened with the study of unipolar chest leads and unipolar limb leads The latter advocated by Wilson and his school[3, 4, 5] and subsequently studied by Goldberger[1] are particularly useful for the recognition of the physiologic variants of the electrocardiogram and are now taken routinely

The hearts of normal subjects may be in different positions within the chest Accordingly a greater or lesser part of either ventricle may face one or the other shoulder or the left leg Therefore tracings recorded by means of the unipolar limb leads explain the cause of several physiologic variants which are necessarily followed by variants of the tracings in the standard limb leads

Several types of rotations of the heart have been recognized Those around the *anteroposterior axis* cause vertical semivertical intermediate semihorizontal and horizontal types of rotation Those around the *longitudinal axis* cause clockwise and counterclockwise rotations Those around the *transverse axis* cause forward and backward rotation of the apex Certain

displacements around one of these axes are usually accompanied by displacement around another thereby limiting the number of possibilities Figures 125 and 126 illustrate the typical variations of the ecg on account of vertical and horizontal positions Tables 11 and 17 explain the same patterns taking into consideration also possible rotations around other axes Judgement on whether the heart is vertical or horizontal can be drawn by comparing aVL with aVF, or Leads 1 with 3 as shown by Fig 127 It can be determined more accurately by using the sketch of Fig 120

TABLE 11 ELECTROCARDIOGRAPH FINDINGS AVERAGE VERTICAL HEART

Lead	*QRS*	*T*	*Explanation*
V1	rS	Sometimes downward	Because of clockwise rotation around the longitudinal axis the right ventricle becomes more anterior therefore V3 & sometimes V4 show a right ventricular pattern (rS)
V2–V3	rS	Upright	
V4	rS or qR	Upright	
V5–V6	qR or qRs	Upright	Leads V5 V6 face the epicardial surface of the left ventricle
aVR	rS	Downward	The right arm lead faces the cavity of the right ventricle
aVL*	*QS*	*Downward*	The left arm lead faces the cavity of the left ventricle
aVF	qR or qRs	Upright	The left leg lead faces the epicardial surface of the left ventricle and the tracing resembles that of V5
VEs 45–55	qR	Upright	A left ventricular pattern is revealed by low esophageal leads and the tracing resembles that of V5

In this and in the following tables the abnormal findings are in italic

The position of the average vertical heart usually includes some degree of clockwise rotation around the longitudinal axis (if a more severe rotation see Table 12 if backward rotation of apex see Table 13)

* aVL may face the right ventricular cavity because of clockwise rotation then an rS pattern with a downward T wave is present

The low esophageal leads give a pattern which is similar to that of aVF Thus in the vertical heart they present a left ventricular pattern, in the horizontal heart a right ventricular pattern It should be emphasized that correlation between anatomic shifts and rotations and electrocardiographic rotations is only approximate and sometimes nonexistent

The average vertical heart has nearly always some degree of clockwise rotation around its longitudinal axis Therefore the right ventricle becomes more anterior, V3 and occasionally V4 may show a right ventricular pattern rS The unipolar lead of the right shoulder may present a QS complex and an inverted T wave because it 'faces the cavity of the left ventricle (Table 11 Fig 125) If a severe clockwise rotation is present the vertical heart presents

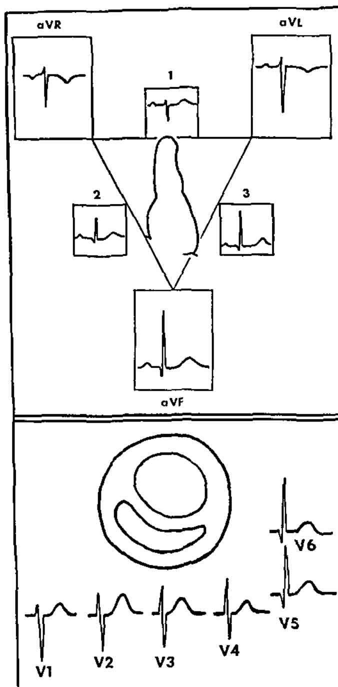


FIG 125 Sketch of the electrocardiogram in the average vertical heart Unipolar limb leads standard leads (smaller scale) and unipolar chest leads

TABLE 12 ELECTROCARDIOGRAPHIC FINDINGS VERTICAL HEART WITH MARKED CLOCKWISE ROTATION AROUND THE LONGITUDINAL AXIS

Lead	*QRS*	*T*	*Explanation*
V1	QR	Sometimes downward	Because of marked clockwise rotation around the longitudinal axis the right ventricle becomes frankly anterior there fore leads V2–V5 (and sometimes V6) show a right ventricular pattern (rS)
V2–V5	qR	Upright	
V6	rS or qR	Upright	
aVR	rS	Downward	The right arm lead faces the back of the heart.
aVL	rS	Upright	The left arm lead faces the epicardial sur face of the right ventricle and its pattern resembles that of V2
aVF	QR	Upright	The left leg lead faces the epicardial surface of the left ventricle & its pattern re sembles that of V5

a right ventricular pattern not only in V3 and V4 but also in V5, and even in V6 (Table 12) In all types of vertical heart the main direction of QRS in aVL is directed downwards in aVF, upward Mnemonically they look like two hands directed toward each other (Fig 126) The same is true for leads 1 and 3

The average horizontal heart is usually accompanied by some degree of counterclockwise rotation around the lon gitudinal axis (Table 14) Therefore leads V4 to V6 face the epicardial surface of the left ventricle and have a qR pattern (Fig 127) A horizontal heart may have a marked counterclockwise rotation around the longitudinal axis (Table 15), though this is seldom true in normal subjects If this occurs all chest leads V1 to V6 have a qR pattern because they 'face the epi cardial surface of the left ventricle and the unipolar lead of the left shoulder has an inverted T An important differential point between this type of physiologically horizontal heart and one with left ventric ular hypertrophy is that in the latter the downward T in aVL is associated with de pressed S T wide QRS and inverted T in the chest leads

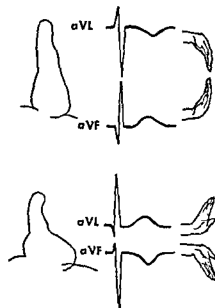


Fig 126 Mnemonic rule for the direction of the QRS in the hori zontal and the vertical hearts aVL and aVF (or 1 and 3)

Forward rotation of the apex is rare in a normal subject If present, V1 and

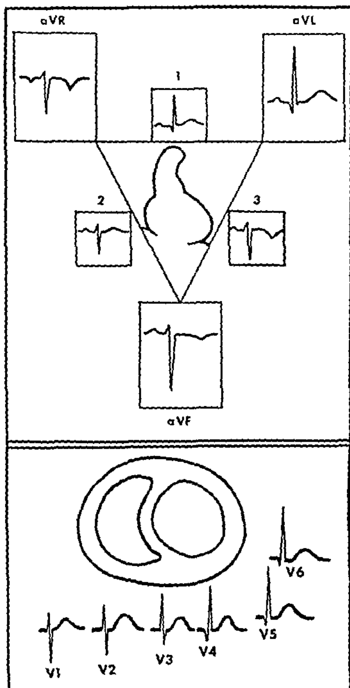


FIG 127 Sketch of the electrocardiogram in the average horizontal heart (see Fig 125)

TABLE 13 ELECTROCARDIOGRAPHIC FINDINGS VERTICAL HEART WITH BACKWARD ROTATION OF THE APEX

Lead	*QRS*	*T*	*Explanation*
VI–V4	rS	Sometimes Downward in V1	Because of slight clockwise rotation V3 and V4 tend to show a right ventricular pattern similar to that of V1
V5–V6	qR	Upright	Leads V5–V6 face the epicardial surface of the left ventricle and their pattern resembles that of V5
aVR	QR	Downward	Both right and left arm leads tend to face the back of the heart
aVL	*QR*	*Downward*	
aVF	rS	Upright	The left leg lead faces the epicardial surface of the right ventricle and its pattern resembles that of V1

V2 have a right ventricular pattern, aVL has an upright T (but it may have an inverted T if there is counterclockwise rotation) (Table 16)

Marked clockwise rotation around the longitudinal axis causes the right ventricle to become anterior Then all chest leads may show a right ventricular pattern rS (Table 17)

In all types of horizontal heart the main direction of QRS in aVL is directed upwards in aVF, downwards, the same is true for leads 1 and 3, mnemonically they look like two hands directed away from each other (Fig 127)

TABLE 14 ELECTROCARDIOGRAPHIC FINDINGS AVERAGE HORIZONTAL HEART*

Lead	*QRS*	*T*	*Explanation*
V1–V3	rS	Sometimes downward in V1	These leads face the epicardial surface of the right ventricle
V4–V3	qR	Upright	These leads face the epicardial surface of the left ventricle
aVR	rS	Downward	The right arm lead faces the right ventricular cavity
aVL	qR	Upright	The left arm lead faces the epicardial surface of the left ventricle and its pattern resembles that of V5
aVF	rS	Upright	The left leg lead faces the epicardial surface of the right ventricle & its pattern resembles that of VI
VEs 45–55	rS	Upright	A right ventricular pattern is revealed by low esophageal leads

* The position of the average horizontal heart usually includes some degree of counterclockwise rotation around the longitudinal axis (if more severe rotation see Table 15 if forward rotation of apex see Table 16 if clockwise rotation see Table 17)

TABLE 15 ELECTROCARDIOGRAPHIC FINDINGS HORIZONTAL HEART WITH MARKED COUNTER CLOCKWISE ROTATION AROUND THE LONGITUDINAL AXIS

Lead	*QRS*	*T*	*Explanation*
V1–V6	qR	Upright	These leads face the epicardial surface of the left ventricle
aVR	rS or QS	Downward	According to whether the right arm lead *faces the right or the left ventricular* cavity a right or left ventricular pattern is recorded
aVL	qR	Downward	The left arm lead faces the posterior epicardial surface of the left ventricle & its pattern is similar to that of V5
aVF	rS	Upright	The left leg lead faces the epicardial surface of the right ventricle and its pattern is similar to that of V1 in a normal heart without rotation

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TABLE 16 ELECTROCARDIOGRAPHIC FINDINGS HORIZONTAL HEART WITH MARKED FORWARD ROTATION OF THE APEX

Lead	*QRS*	*T*	*Explanation*
V1	rS	Sometimes downward	This lead faces the epicardial surface of the right ventricle
V2	rS or qR	Upright	These leads face the epicardial surface of the left ventricle because of counter clockwise rotation (usually assoc)
V3–V6	qR	Upright	
aVR	qR	Downward	The right arm lead faces the posterior surface of the heart
aVL	qR	Upright	The left arm lead faces the epicardial surface of the left ventricle & its pattern resembles that of V1
aVF	rS	Upright	The left leg lead faces the epicardial surface of the right ventricle & its pattern resembles that of V1

TABLE 17 ELECTROCARDIOGRAPHIC FINDINGS HORIZONTAL HEART WITH MARKED CLOCKWISE ROTATION AROUND THE LONGITUDINAL AXIS

Lead	*QRS*	*T*	*Explanation*
V1	rS	Sometimes downward	Because of marked clockwise rotation the right ventricle becomes frankly anterior and sometimes all the precordial leads show a right ventricular pattern (rS)
V2–V5	rS	Upright	
V6	rS or qR	Upright	
aVR	QR	Downward	The right arm lead faces the back of the heart
aVL	qR	Upright	The left arm lead faces the epicardial surface of the left ventricle and has a pattern similar to that of V6 in a normal heart
aVF	rS	Upright	The left leg lead faces the epicardial surface of the right ventricle and has a pattern similar to that of V1

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C H A P T E R 3 4

Tracings of the Fetal Heart

ELECTROCARDIOGRAM

History

Cremer[5] in 1906 and Foa in 1911 recorded the first electrocardiograms of the human fetus towards the end of pregnancy Maekawa and Toyoshima[13] later obtained tracings by means of a string galvanometer and a preamplifier After a series of further observations in individual cases systematic studies were made by Heard and co workers[9] Strassman and co workers [3] [4] Dressler and Moskowitz[6] Mann and Bernstein[14] Ward and Kennedy[25] and Blondheim

Technic

The standard electrocardiograph is unable to pick up the currents of the fetal heart because the potentials reaching the surface of the mother s body are extremely weak Either an electroencephalograph or an electrocardiograph with a preamplifier is necessary for the study Even though vaginal or rectal leads have been recommended and may be used with good results the abdominal leads are usually sufficient Strassman [3] [4] and Blondheim described several bipolar abdominal leads

Wilson s central terminal can be used as an indifferent electrode while

the exploring electrode (a round chest electrode) is applied successively over several points of the midline or over one of the upper quadrants The instrument should be calibrated at a sensitivity of 10 cm for 1mV On the other hand the tracings can be taken more easily by using two abdominal electrodes This technic records smaller deflections for the maternal electrocardiogram and there is less influence by AC induction

Tracings Obtained

The fetal electrocardiogram can be recorded after the fourth month of pregnancy but the frequency of positive tracings increases gradually until a successful result is the rule after the sixth month [24]

The fetal electrocardiogram consists of small deflections—either single sharp spikes or diphasic complexes—superimposed over the tracing of the

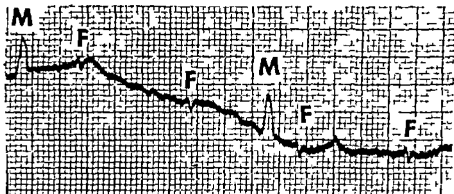


FIG 128 Electrocardiogram of a fetus at term The small fetal complexes (*F*) are visible between those of the mother (*M*) (amplifier, two abdominal electrodes)

maternal electrocardiogram The T wave is frequently visible as a small rounded wave which is either positive or diphasic The rapid complex lasts from 0 02 to 0 04 second (Fig 128)

Fetal heart rate is extremely variable from hour to hour and arrhythmias are frequent The latter have been identified as sinus arrhythmias and premature contractions mostly ventricular Gross irregularities due to atrial fibrillation were noted by Hyman,[11] complete or incomplete a v block was observed by Geiger and Hines[8] and by Plant and Steven [18] Spontaneous variations of heart rate have been noted following fetal movements palpation of the maternal abdomen fasting or hemorrhage of the mother asphyxia of the fetus, and various other conditions

The normal heart rate of the fetus varies between 140 and 150 [14]

Conclusions

Fetal electrocardiography has been used for (1) diagnosis of pregnancy (2) diagnosis of fetal arrhythmias (3) diagnosis of fetal life The last is the most important application and in this respect the method may be unique

FETAL PHONOCARDIOGRAM

History

The existence of fetal heart sounds was detected in 1818 by Mayor[15] The first phonocardiograms of the fetal heart were recorded by Hofbauer and Weiss[10] in 1908 and by Beruti[1] in 1923 Sampson MacCalla and Kerr[19] transcribed the fetal souffle Hyman[11] studied the arrhythmias of the fetal heart Peralta Ramos[16] made the prenatal diagnosis of congenital heart block by phonocardiography Smith and Herwert[21] Lian and Smith[2] and Pereira[17] published subsequent contributions Transcription of fetal sounds on records transmission of fetal sounds to audiences and observation by means of an oscilloscope has been done[3]

Technic

The phonocardiographic technique is similar to that employed for recording the heart sounds of an adult (p 35) A stethoscopic microphone with a large chest piece is used with a long rubber strap around the abdomen of the mother The phonocardiogram of the fetus is recorded together with the maternal electrocardiogram If the fetal sounds have been already detected with the stethoscope the microphone is placed over the area of best auscultation Otherwise several tracings are taken with the microphone over different areas of the abdomen Inspection of the tracing (and frequently also amplified auscultation) reveal heart sounds with a rate much faster than that of the maternal heart

Interpretation

A fourth (atrial) sound made of one or two slow vibrations is frequently recorded It starts about 0 04–0 05 second before the first fetal sound A brief pause may exist between the fourth and the subsequent first sound

The first sound complex starts about 0 02 second after the Q wave of the fetal ecg It is made of 3–5 vibrations and lasts from 0 03 to 0 10 second Lian and Golblin[1] were the first to note that it is frequently divided in two groups of vibrations like the first sound of the adult heart

The second sound complex is made of two to four vibrations having a higher pitch than those of the first sound Its amplitude may be greater or

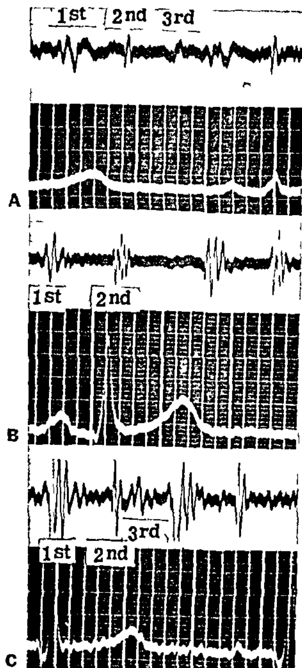


FIG 129 Fetal phonocardiograms and maternal electrocardiograms *A*, 6 month-old fetus *B*, 9 month-old fetus (same child as *A*) *C* 9 month old fetus

smaller than, or equal to, that of the first sound It lasts from 0 02 to 0 07 second Occasional splitting of this sound is not uncommon

It has been claimed that the third sound is never recorded Actually it may be present *about 0 12 second after the* second sound as in Fig 129A Fetal systole lasts about 0 14 second (minimum observed 0 11, maximum 0 22) Fetal diastole varies between 0 14 (rate 230) and 0 28 (rate 145) In one of our cases systole was 0 14 at 6 months and 0 22 at 9 months (Fig 129)

A systolic murmur is frequently observed It is probably caused by the shunts which normally exist during fetal life (foramen ovale ductus arteriosus) However the rapid circulation of the blood in the large arteries may be a contributing factor A suspicion of ventricular septal defect may arise if the murmur is loud and even more if it is associated with bradycardia *Prenatal* diagnosis of septal defect has been made by Peralta Romas[16] and by Smith [9] An *umbilical souffle* having the same rate as the fetal heart and different from the *uterine souffle* has been recorded by Pereira[17] and is an occasional finding

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CHAPTER 35

Tracings in the Child

HEART RATE

The heart rate in the child decreases gradually from birth to puberty This decrease seems to be connected with the increasing length of cardiac bundles and the increasing weight of the heart[3] and not with variations in the tonus of the vagus nerve (Mautner and co workers[9]) Table 18 from data by Pfaundler and Schlossmann[11] and Holt and McIntosh [6] compares heart rates body weights and heart weights showing a fairly close correlation

TABLE 18 HEART WEIGHT AND RATE

Age (yr)	*Body weight (Kg)*	*Heart weight (gm)*	*Heart rate (per minute)*
Newborn	3 3	23	135
1	9 5	42	115
2	12	50	105
5	16	65	90
10	26	103	78
15	44	163	68

A variety of morphologic characteristics of the heart of infants is responsible for several peculiarities of the tracings especially the electrocardiogram They were summarized by Battro and Mendy[3] as follows

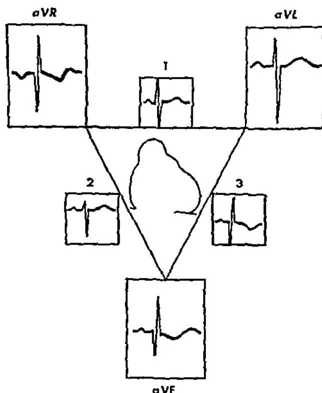


FIG 130 Scheme of the electrocardiogram of a 2 month-old infant

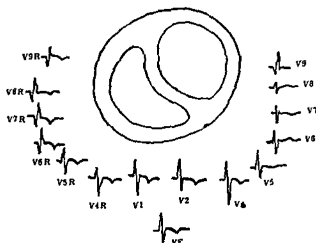


FIG 131 Conventional chest leads and right side chest leads in a 2 month-old infant

1 The heart mass of the infant is smaller than that of the adult but the heart-weight/body weight ratio is greater The heart has a globular shape

2 The various diameters of the chest (anteroposterior, transverse, and vertical) are about equal, in contrast with the adult chest which has a smaller anteroposterior diameter

3 The heart is nearer to the thoracic wall than in the adult and there is little or no pulmonary *parenchyma between* heart and chest wall

4 The chest wall is thinner than in the adult

5 The diaphragm is higher *and the heart is more* horizontal than in most adults

6 At birth size and thickness of the two ventricles are about equal in the older children the left ventricle has a greater thickness than the right

ELECTROCARDIOGRAM

The electrocardiogram of the child has been studied by Nicolai and Funaro [10] Master *et al* [8,1] Rosenblum and Sampson [11]

Robinow *et al* [13] Groedel *et al*,[4 5] Ash Battro and Mendy [3] and Massell and co workers [1]

At birth the standard leads reveal right axis deviation the unipolar limb leads indicate a vertical heart the chest leads reveal a predominance of the R over the S waves The T waves are inverted diphasic or isoelectric from V1 to V4 V5 and even V6 (Figs 130 and 131) As the child grows older a tendency to diphasism of the complex with actual presence of S is noted At the age of 10 an adult type of electrocardiogram is usually recorded and the T wave is usually upright in V4

PHONOCARDIOGRAM

The phonocardiogram of the child below 4 years of age frequently presents four sounds Figures relating to the sound tracings in this period of life have been published by the author and coworkers[7] and can be found in Table 3 (p 46) An example of infantile phonocardiogram is presented in Fig 24 (p 45)

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CHAPTER 36

Tracings in Pregnancy and Puerperium

Pregnancy is accompanied by numerous and important modifications of cardiovascular dynamics which are revealed by changes of the various tracings The heart becomes more horizontal on account of raising of the diaphragm and of lumbar lordosis The increased blood volume causes a slight dilatation of the various chambers and a more marked dilatation of the large vessels especially the pulmonary artery Innocent murmurs are common Among them the most frequently observed is a systolic murmur at the base with maximum over the second left interspace probably caused by trigonoidation of the pulmonic and aortic orifices with relative stenosis [8] An apical systolic murmur and one or more extra sounds in diastole (third and fourth sounds—so called gallop sounds) may simulate the auscultatory findings of rheumatic heart disease [8] However this finding is less common than that of a basal systolic murmur If there is rheumatic heart disease with mitral lesions the murmurs become louder during pregnancy An accurate diagnosis of heart disease during pregnancy is rendered difficult by addition of myocardial murmurs to those of the valvular lesions

ELECTROCARDIOGRAM

The ecg changes have been studied by Carr and Palmer [1] Feldman and Hill [3] Gammeltoft [4] Hollander and Crawford [5] Jensen and Norgaard [6] Landt and Benjamin [7] and Zatuchni [9] With advancing pregnancy the electrical position of the heart either remains the same or becomes more horizontal

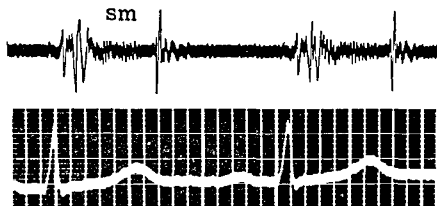


Fig 132 Normal maternal heart at 6 months of pregnancy Systolic murmur (*sm*) over the pulmonary artery

4th 1st 2nd 3rd 4th

A

B

Fig 133 Pregnant woman with minimal valvular lesions The murmurs become louder during pregnancy *A* At 5 months of pregnancy Loud systolic murmur and triple rhythm at apex *B* After delivery (faint systolic murmur)

Late in pregnancy, it usually is in the intermediate position Following delivery the heart returns to the previous position

The variations of the electrical axis during pregnancy may be considerable (in one case of Zatuchni[9] the axis varied from 86 to 144 5 degrees) A rotation of the heart around its anteroposterior axis was noted in the majority of cases during pregnancy[9]

The amplitude of the R wave and the duration of the intrinsicoid deflection in the left precordial leads do not vary during pregnancy[9] The amplitude of the T wave during pregnancy is usually within normal limits but changes in the height of this wave have been noted following delivery Decrease of T in V1 V2 and increase in V4 V6[9] have been attributed to systemic biochemical changes and not to myocardial disease

PHONOCARDIOGRAM

The phonocardiogram confirms the presence of murmurs already observed by auscultation (Fig 132) Basal systolic murmurs have a diamond shaped appearance apical systolic murmurs a decrescendo type (Fig 34) The vibrations of the third and fourth sound are frequently large Figure 133 illustrates a case with moderate valvular lesions where the murmurs were severely increased by pregnancy

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CHAPTER 37

Tracings in the Aged

The heart of the aged frequently presents valvular, myocardial and arterial lesions which determine abnormalities in the electrocardiogram phonocardiogram electrokymogram etc However it should be kept in mind that a surprising number of old persons who reach the seventh or eighth decade have a clinically normal heart

Many old people have slight abnormalities of the electrocardiogram which are merely the result of senescence like lower voltage low T waves slight prolongation of QRS or *P R*[1] However about one fourth of the apparently normal old persons present more severe electrocardiographic abnormalities indicating myocardial damage[2] These consist of the various changes resulting from coronary heart disease with myocardial lesions (pp 419 and 454) Among them, atrial fibrillation bundle branch block depression or elevation of S-T, and inversion of T have been described[3]

The phonocardiogram may reveal evidence of valvular or myocardial damage or atherosclerosis of the aorta (pp 432 and 469) However one should keep in mind that persons between 85 and 95 may present an absolutely normal phonocardiogram as proven by studies of the author (Fig 26 p 47)[4]

In older individuals with a presumably normal heart the following changes *of the ballistocardiogram have been found*[2]

1 Increased amplitude of respiratory variations

2 Increased amplitude of the H wave which may even exceed the J
3 Decreased amplitude of the I wave which may only reach the base line
4 Decreased amplitude frequent slurring and late peak of J
5 Deep and early K this wave may precede the second heart sound
6 Large L wave

These variations are particularly apparent in the expiratory phase of normal respiration

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CHAPTER 38

Tracings in Various Mammals

HEART RATE

The heart rate varies inversely with the heart weight regardless of the species and of the fact that the heart weight body weight ratio varies from species to species[11] This fact is even more striking when comparison is made between house animals and hunting or racing animals Hunting dogs and race horses have much lower heart rates than other animals of the same species partly because of increased heart size and partly on account of increased tonus of the vagus nerve As an exception to the general rule the horse has a rate similar to that of the elephant in spite of the smaller heart

Comparison of two species having a ratio of heart weight greater than 1 1000 reveals that the ratio of heart rates is less than 10 1 This might indicate that the different rates are proportional to the different lengths of certain bundles and not to the weight of the heart or to specific properties of the different muscle cells[11]

Comparative data on heart rate as well as on other properties of the animal heart can be found in Dukes[4] and are listed in Table 19

ELECTROCARDIOGRAM

History

Many electrocardiographic studies were made in animals either in order to perfect electrocardiographic technic or to solve problems of cardiac

TABLE 19 THE ELECTROCARDIOGRAM OF VARIOUS MAMMALS

Animal	*Rate*	*P*	*P R interval*	*QRS*	*Q T interval*	*T*
Mouse	650–900	Upright	0 05	0 015	0 06	Upright small
Rat	380–520	Small 0 04	0 05	0 02	0 08	Upright rounded
Guinea pig	200–325	Sharp 0 04	0 06–0 07	0 03	0 12	Upright sharp
Rabbit	170–280	Possibly Inverted	0 06–0 10	0 03	0 18	Possibly inverted
Cat	110–130	0 04	0 08	0 03	0 18	Upright rounded
Dog	70–120	0 06–0 08	0 10–0 13	0 03–0 05	0 20–0.24	Diphasic sharp
Goat	70–120	Double 0 08 diphasic	0 12–0 14	0 05	0 26–0 32	Monophasic
Sheep	52–100	Double low 0 08	0 08	0 04	0 24	Monophasic
Pig	60–90	Simple very high	0 12–0 14	0 06	0 22–0 28	Late sharp diphasic
Donkey	60–80	Triphasic small 0 08	0 20	0 06	0 40–0 44	Diphasic
Cow	60–80	0 10–0 30 diphasic or triphasic	0 16–0 30	0 08–0 12 deformed	0 36–0 44	Sharp high often diphasic
Bull	36–60	0 10 simple	0 16–0 20	0 08–0 10	0 36–0 40	Sharp upright or diphasic
Horse	24–50	0 15–0 22 double or multiple	0 35–0 42	0 08–0 12	0 40–0 60	Sharp often diphasic
Elephant	24–53	Double 0 12–0 20	0 28–0 35	0 12–0 18	0 52–0 70	Diphasic

physiology Little attention was often paid to the special properties of the heart of the animal under study Moreover most of the animals were under anesthesia so that data concerning heart rates were not reliable Only a few recent studies have been conducted with unipolar leads

Horse Electrocardiograms were taken by Einthoven[9] Waller[7] Kahn[1] Noerr[20] Dukes and Batt[8] Charton and Minot[5] by the author and his co workers[18] and by Detweiler[29]

Elephant Following studies of Benedict[3] detailed electrocardiographic studies were published by White Jenks and Benedict[8]

Donkey The only study is that of the author and his co workers[18]

Ox The electrocardiogram was studied by Noerr[1] Alfredson and Sykes[1] Barnes and co workers and by the author[18]

Pig The electrocardiogram of this animal has been studied by Haussman[11] and by the author[18]

Sheep and Goat Strohmaier[illegible] and the author[18] studied the electrocardiogram of these species

Dog Electrocardiograms of mongrel dogs were recorded by Katz and

co workers [13] Hafkesbring and MacCalmont,[10] Lalich and co workers,[14] Mainzer and Krause[10] and by the author and co workers [18] Tracings of normal beagle dogs have been studied by Petersen and co workers [3]

Cat The electrocardiogram of the cat was studied by Schinzel[23] and by the author [10]

Rabbit and Guinea Pig Electrocardiographic studies were published by Schinzel Levine,[1] and by the author [18]

Rat and Mouse Electrocardiographic studies were published by Schinzel Oppenheimer [22] the author (rat only) [18] Lombard [30] and Rappaport and Rappaport [4] The last study was made with a specially built instrument

Monkey The electrocardiogram of these animals was studied by Boruttau[4] and found identical to that of man

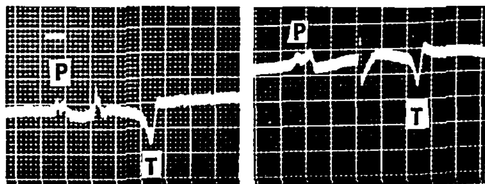


Fig 134 Electrocardiogram of two normal horses Notched P waves long P R intervals

Technic

Small animals can be tied on laboratory tables and studied in the supine position Larger animals should be studied standing The rat has to be immobilized by means of holding devices

Following shaving of the hair electrode jelly is applied to the roots of the four limbs and the usual electrodes are employed If the electrical resistance of the skin is high as in some large animals needle electrodes can be used In the elephant special electrodes on which the soft pads of the feet were placed have been used [8]

For the chest leads the author has used three electrodes one at the right side of the sternum one over the sternum and one at the left of the sternum These should be called V1 V2 and V3 and connected with the central terminal In large animals the three electrodes should be placed about 3 to 5 inches below the axillae on account of the high position of the heart

Electrocardiographic Data

The main data have been listed in Table 19 The heart rates vary between a maximum of 900 (mouse) and a minimum of 24 (horse and elephant) the P R interval between 0 05 (rat and mouse) and 0 42 (horse) the duration of QRS between 0 015 (mouse) and 0 18 (elephant) the Q T interval between 0 06 (mouse) and 0 70 (elephant) Electrocardiograms of various animals are presented in Figs 134 to 137

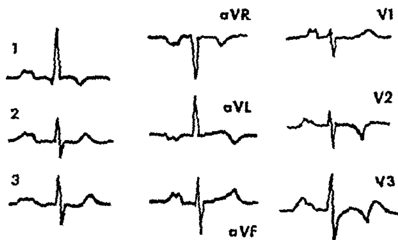


Fig 135 Scheme of the electrocardiogram of the horse in the standard and unipolar limb leads and in the chest leads

The electrocardiogram of the horse is among the most peculiar tracings It has a bifid or multiple P wave and a P R interval which is long even in comparison with the average size of the heart (Figs 134 and 135) The interval between end of the T wave and the second heart sound may reach even 0 12 second (Fig 139) Studies by the author[16] in the horse dog and cat and of Petersen *et al* [3] in the beagle dog by means of unipolar limb and chest leads reveal that the heart of the various animals is in a vertical or semivertical position (Figs 135 and 137) AVL frequently presents inverted P and T waves

PHONOCARDIOGRAM

History

Heart sounds of horses have been recorded by Charton and co workers [6] Systematic studies of the author and co workers were made in normal animals[18] and in animals under the influence of various drugs [17]

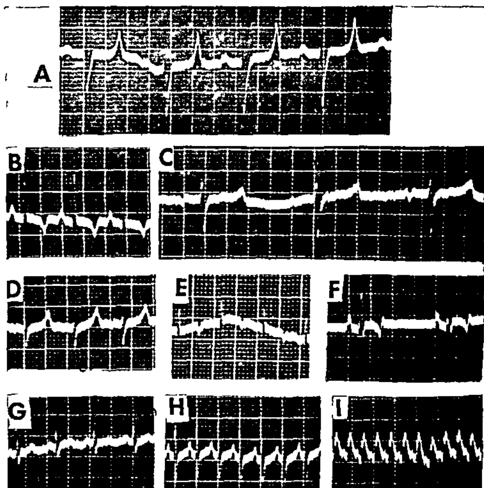


FIG 136 Electrocardiograms in various species *A*, bull *B* pig *C* donkey *D* goat *E* sheep *F* dog (anesthesized), *G* cat (anesthesized) *H*, rabbit *I* rat

Technic

The technic is identical to that used in humans A stethoscopic microphone with a large bell should be used It is placed at the right or the left of the sternum and held in place by means of a long rubber strap Reference of sound and murmurs to certain valves is only tentative Tricuspid sounds and murmurs are recorded best at the right of the sternum, mitral sounds and murmurs, at the left, aortic and pulmonic sounds and murmurs are recorded best at the base

Phonocardiographic Data

The multiplicity of data renders necessary its summarization (Table 20 p 300) The heart sounds increase in duration from the small to the large animals as follows

First sound from 0 03 (guinea pig) to 0 24 (certain horses)
Second sound from 0 02 (guinea pig) to 0 12 (certain donkeys)
The third sound is sometimes present in the horse and donkey the fourth (atrial) sound is frequently present in the guinea pig sometimes in the dog pig and horse

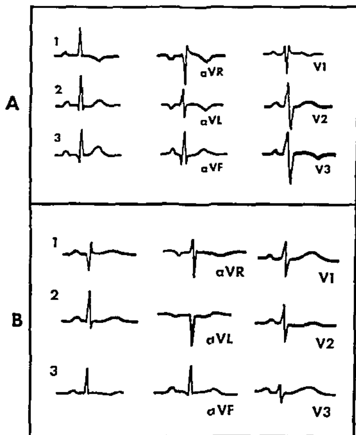


FIG 137 Unipolar standard and chest leads *A* in a normal dog *B* in a normal cat

The duration of ventricular systole (distance between first and second sound) varies from 0 12 in the guinea pig to 0 62 in certain horses

Guinea pigs and rabbits frequently have respiratory murmurs superimposed over the cardiac sounds on account of similar cardiac and respiratory rates (Fig 138) *The horse frequently exhibits some low pitched vibrations* about 0 2 second after the second sound (Figure 139)

Murmurs due to valvular damage are clearly recognizable and tend to be low pitched and musical in large animals (Figs 140 and 141)

TABLE 20 HEART SOUNDS OF VARIOUS MAMMALS

Animal	Fourth (atrial) sound	First sound	Second sound	Distance between first and second sounds (ventricular systole)	Third sound	Murmurs
Guinea pig	present high	3–4 vibrations 0 03	2–3 vibrations 0 02–0 03	0 12	—	due to respiration
Rabbit	present high	short slow vibrations 0 04	short slow vibrations 0 04	0 18	—	due to respiration in systole and diastole
Cat	absent	pure 0 05	very short 0 04	0 18	—	absent
Dog	sometimes present	pure 0 08	pure 0 06	0 20–0 24	—	absent
Goat	absent	prolonged by short vibrations 0 07	pure 0 06	0 28	—	absent
Sheep	absent	prolonged 0 07	prolonged 0 04	0 22	—	absent
Pig	present	prolonged 0 12–0 20	pure 0 04–0 06	0 28	—	absent
Cow	absent	pure 0 12–0 20	pure	0 52	—	absent
& Bull	present	pure 0 12–0 20	sometimes split 0 04–0 06	0 32	—	absent
Horse	absent	pure 0 15–0 24	pure 0 08	0 52–0 62	sometimes present	some slow vibrations after second sound 0 20
Pony	sometimes present	8–9 vibrations 0 12	4–5 vibrations 0 08–0 10	0 60	sometimes present	absent
Donkey	absent	4–5 vibrations 0 14–0 16	3–4 vibrations 0 07–0 12	0 44–0 58	present	absent

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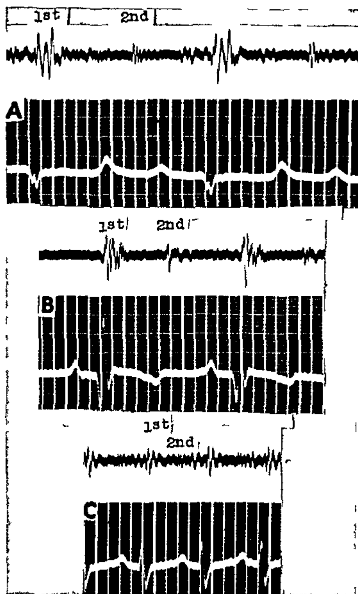


FIG 138 Phonocardiograms (and electrocardiograms) of various species *A* bull *B* dog *C* rabbit

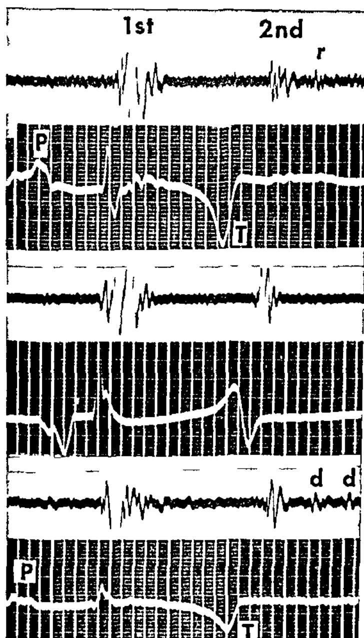


FIG 139 Phonocardiograms of three normal horses *r* indicates an early-diastolic rumble of brief duration

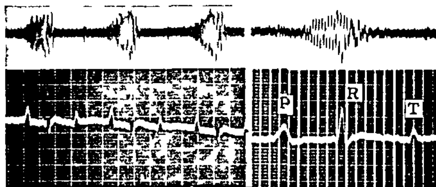


FIG 140 Presystolic murmur (*pm*) in a horse with valvular lesions *Left* rapid film speed *right* slow film speed

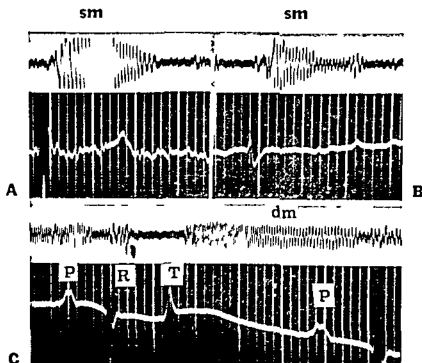


FIG 141 Musical murmurs of horses with valvular lesions *A* and *B* systolic murmur (*sm*) in two different animals *C* diastolic murmur (*dm*) in a third horse

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CHAPTER 39

Modifications of Tracings Caused by Drugs and Physiopathologic Variants

Various physiologic and pathologic processes as well as many commonly used drugs can appreciably modify cardiovascular dynamics Changes of the cardiac and respiratory rates arterial blood pressure and venous return result in modifications of the various tracings Ectopic rhythms and variations of a v conduction are not unusual Apart from the result of these variations specific changes of the electrocardiogram follow the use of certain drugs The variations of the electrocardiogram have been summarized in Table 21, partly made from data of Graybiel and White [2]

TABLE 21 EFFECTS ON THE ELECTROCARDIOGRAM OF DRUGS AND PHYSIOLOGIC PROCESSES

ACIDOSIS
Rate Slight increase
There may be depression of the S-T segments lowering of the T waves prolongation of the Q T interval and rarely atrial fibrillation factors other than the acidosis may play a role

ADRENALIN
Rate Slight to moderate increase
S T May be depressed
Given intravenously adrenalin may cause T wave inversion in lead 2 In persons with marked coronary insufficiency adrenalin may cause depression of S T and lowering or inversion of T

ALKALOSIS
Rate Slight increase
P Little or no change
A marked degree of alkalosis usually produces sagging of the S-T segments and lowering or even inversion of T (as from digitalis) and lengthening of the Q T interval Overventilation acts by producing alkalosis

TABLE 21 (*Continued*)

ATROPINE
Rate Increased
QRS Amplitude may become reduced
T May become lowered or even slightly inverted
With increasing age the effect progressively diminishes

BARBITURATES
Rate Slight decrease
Through the effect of the drug on the central nervous system sinus arrhythmia may be abolished and there may be a lessened tendency toward cardiac arrhythmia

BENZEDRINE SULFATE
Rate May decrease
S T May become depressed
T May become taller
The effect is variable depending upon dosage and patient

CALCIUM (hypercalcemia)
Rate Decreased
T May become lowered or inverted
Q T Shortened

CALCIUM (Hypocalcemia)
Q T Lengthened

CARBON MONOXIDE
Rate Various
P Slight changes
QRS Voltage may become lower
S T May become depressed
Q T May be lengthened
There are no significant changes associated with mild poisoning

DIGITALIS
Rate May be decreased rarely increased
P R Lengthened
S T Depressed or concave
T Usually lowered or even inverted
Q T May be shortened
The effects vary with dosage age of patient and presence of heart disease or other disorder

EMETINE HYDROCHLORIDE
S T May become depressed
T May become lowered or even inverted
The marked changes are observed in instances of drug toxicity

ERGOTAMINE TARTRATE
Rate May be decreased
T May become taller

EXERCISE
Rate Increased
QRS Tendency toward right axis shift
S T May become depressed
T May become taller
The effect is variable and depends on the amount of exercise individual variation and presence of disease

FRIGHT
Rate Increased then slowed
P May vary in shape
S T May become slightly depressed
T May become lowered or even inverted
The electrocardiographic changes may be significant but are temporary

GLUCOSE (Hypoglycemia)
Rate Increased
S T May become depressed
T May become lowered or even inverted
Changes are more likely to appear in persons with heart disease

HEAT (Hyperpyrexia)
Rate Increased
S T May become slightly depressed
T May become lower
The changes are usually slight but they are marked occasionally in cases of hyperpyrexia

INSULIN
S T May become depressed
T May become lower or inverted
The changes are due to the lowering of the blood sugar Insulin may abolish electrocardiographic changes in diabetic acidosis

IPECAC
Rate May be decreased
May restore normal rhythm in the presence of paroxysmal auricular tachycardia

MECHOLYL
Rate Usually increased
T May become lower
In the presence of certain arrhythmias mecholyl may restore normal rhythm The effects are not only those simulating vagal stimulation

MORPHINE
Rate May decrease slightly
Even large doses produce no significant changes

NITRITES
Rate Increased
The increase in heart rate may abolish sinus arrhythmia and premature beats There may be a reversal of S-T and T wave abnormalities as a result of favorable action on heart and circulation Partial a-v block may be abolished

TABLE 21 (*Continued*)

OXYGEN LACK OF (anoxia)
Rate Usually increased
P Amplitude may increase
P R May lengthen
QRS Voltage may be reduced
S T May become depressed
T Lowered or even inverted
The changes parallel the degree of anoxia The administration of 100 per cent oxygen may occasionally cause reversal of certain electrocardiographic abnormalities

POSITION CHANGE FROM SITTING TO LYING
Rate Usually decreased
QRS Slight change in electrical axis
S T May become depressed
T May become taller
Any change in the position of the heart in relation to the thorax may alter the electrocardiogram considerably

POTASSIUM (high blood level)
Rate May be decreased
P R May be lengthened
QRS Voltage may be reduced Bundle branch block may develop
S T May become depressed
Q T Variable
High blood levels (as in potassium poisoning in uremia) depress or paralyze atrial action and produce striking and characteristically high T waves Lesser changes are more common

POTASSIUM (low blood level)
Rate May be increased
P R May be lengthened
QRS May be prolonged Voltage may be reduced
S T Depressed
T Lowered or inverted
Q T Prolonged
The electrocardiographic changes tend to disappear following the administration of potassium

QUINIDINE
P R May be lengthened
QRS Usually no change may cause bundle branch block
S T May become depressed
T May become lower
Q T May become prolonged
Effect variable depending on dosage individual susceptibility presence of heart disease etc May restore normal rhythm in the *presence of ectopic rhythm*

THYROID EXTRACT (in myxedema)
Rate Increased
P May become taller
P R May become shorter
QRS Voltage may be increased
S T Usually no change may become elevated
T Voltage increases considerably from a very low level
Q T May become shorter
All electrocardiographic changes associated with myxedema are usually abolished

THYROID EXTRACT (in excess)
Rate Increased
T May become lower or taller
Ordinary doses administered to healthy persons produce insignificant changes in the electrocardiogram

TOBACCO
Rate May be increased
S T May become slightly lowered
T May become lowered or increased
Excessive use by susceptible persons may cause tachycardia premature beats paroxysmal tachycardia and atrial fibrillation

DIGITALIS

Digitalis has been the object of several important studies from Cohn and Fraser[1] in 1913 to McMillan and Bellet[3] in 1950

The main effects of digitalis are a lowering of the S T segment with a typically oblique line a lower and diphasic T wave an oblique T P line These changes are present in the unipolar and standard limb leads as well as in the chest and esophageal leads Inversion of T is not directly caused by digitalis If however the myocardium was damaged prior to administration of the drug then digitalis may cause inversion of T a more deeply inverted T,

CHAPTER 40

Rheumatic Disease (Rheumatic Fever)

As known this disease is most frequent in infancy and childhood Many cases are not diagnosed during the active stage because the signs and symptoms are minor vague or atypical Together with the evidence supplied by determination of the sedimentation rate and study of the charts of weight temperature and radial pulse the graphic methods supply important data both for the diagnosis and for the evaluation of an inflammatory process of the heart

ELECTROCARDIOGRAM

This tracing is frequently of help for the diagnosis of rheumatic carditis Electrocardiographic changes were found in about 20 per cent of the cases by Bland and co workers[3] Electrocardiograms taken at frequent intervals are even more important than one tracing alone or tracings taken at long inter vals Minor changes unimportant in themselves may become significant if modifications are noted in weekly or monthly check ups They should be accepted as evidence of the evolution of an inflammatory process of the myocardium

Changes of Rate and Rhythm

It is customary to observe *sinus tachycardia* (Figs 143A B C) It is less well known that in some cases *sinus bradycardia* (Fig 143D) or severe

sinus arrhythmia are caused by the rheumatic process It is likely that they are due to reflex vagal stimulation caused by impulses which arise in the myocardial wall The meaning of these changes is revealed by the fact that they disappear as soon as the active stage is over

Premature contractions are common, atrial, nodal or ventricular extra systoles have been noted Their diagnostic importance is limited if unaccompanied by other signs and if they arise from a single focus Multifocal extra systoles on the contrary, indicate widespread myocarditis Atrial fibrillation or flutter paroxysmal tachycardia shifting pacemaker, nodal escapes, s a block or a v block may occur [4, 5] Each of these disturbances is evidence

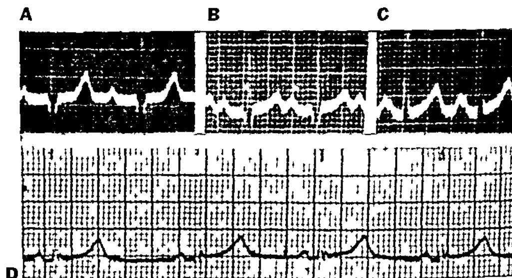


FIG 143 Electrocardiograms in children with acute rheumatic fever *A* Sinus tachycardia P R=0 24 Q Tc=0 44 (QT ratio=1 1) *B* Sinus tachycardia, P R=0 22, Q Tc=0 43 (QT ratio=1 07) *C* Sinus tachycardia Notched P wave P R=0 20 Q Tc=0 46 (QT ratio=1 15) *D*, Sinus bradycardia rate of 56

of severe myocarditis The a v block may indicate localized inflammation of the conducting tissues while the other disturbances are evidence of a more widespread damage

Prolonged P R Interval

Delayed a v conduction may be the only sign of rheumatic carditis (Fig 143A B) This sign is frequently helpful in the diagnosis However, it should be kept in mind that (1) it is not specific (2) it may be absent, and (3) it may be dependent upon reflex vagal stimulation Bland and co workers[3] found this sign in only 20 per cent of their cases P R changes may occur abruptly they may last briefly or become permanent In the evaluation of the P R interval correlation with the age and rate is necessary

Prolongation of the Q T Interval

A prolongation of Q T has been reported as an important sign of rheumatic carditis[5] (Fig 143A B C) Measurement of this interval should be done carefully and the Q T ratio should be determined (p 238) This sign is important when present but is not constant [8]

Changes of the P Wave

The P wave may present broadening notching or increased height or width These changes, influenced by atrial dilatation and hypertrophy cannot be accepted as direct evidence of carditis because they may be due to established fibrosis (older lesions)

Changes of S T and T

Changes of S T are usually connected with pericarditis Isolated changes of T in one or more leads have been observed by the author

Axis Deviation

A rapid and marked shift of the electric axis has been observed in acute rheumatic disease[3] It is more commonly a shift to the right and may be followed by rapid normalization Dilatation of either ventricle possibly related to ventricular strain and failure seems to be the cause of this change

PHONOCARDIOGRAM

The sound tracing of patients with rheumatic disease supplies important data which are of help for confirmation or clarification of the auscultatory findings and also often for establishing a correct diagnosis

The most common findings are

1 A prolonged first sound at the apex

2 A systolic murmur at the apex and midprecordium (Fig 144 A) This is usually a murmur in decrescendo but it may consist of a few vibrations of high pitch and small amplitude during the central phase of systole (sea gull cry type of murmur) (p 55)

3 A split second sound over the pulmonic area (Fig 144)

4 A systolic murmur over the pulmonic area (Fig 144 B C) This is normally diamond shaped and composed of vibrations having a lower pitch than those of the apical murmur

5 An increased loudness of the third (more seldom of the fourth) sound at the apex creating a triple rhythm (older terminology— gallop rhythm)[6, 8] (Fig 144 A)

6 A short and low pitched early diastolic rumble over the midprecordium

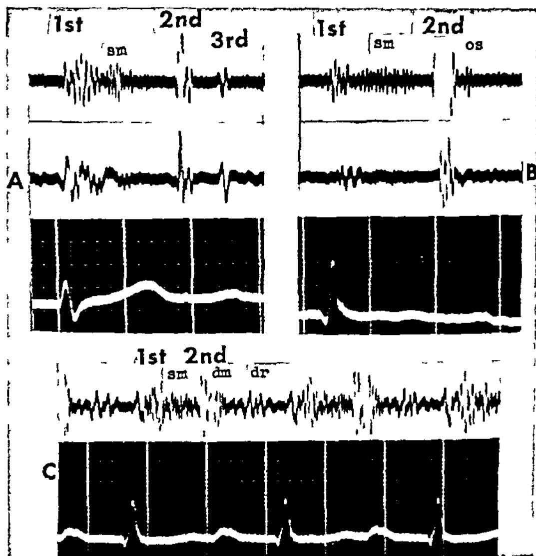


FIG 144 Phonocardiograms in cases of acute rheumatic fever (superimposed tracings) From above logarithmic and stethoscopic phonos ecg
A, Apex Systolic murmur (*sm*) triple rhythm especially revealed by the stethoscopic tracing (*s*)
B Pulmonic area Systolic murmur (*sm*) especially revealed by the logarithmic tracing split second sound opening snap of the mitral valve (*os*) better revealed by the logarithmic tracing
C Pulmonic area Systolic murmur (*sm*) split second sound with a short diastolic murmur (Graham Steell murmur-*dm*) diastolic rumble (*dr*) Stethoscopic tracing

Any or all of these findings may be present and their evaluation is not always easy

The apical systolic murmur is caused by dilatation of the left ventricle and by edema of the mitral valve and of the papillary muscles (relative mitral regurgitation) The midprecordial short *diastolic rumble* is a functional phenomenon caused by dilatation of the left ventricle and increased pressure in the left atrium (relative mitral stenosis) The *pulmonic systolic murmur* is caused by dilatation of the pulmonary artery (relative pulmonic stenosis) The split second sound is due to increased pulmonic pressure The triple rhythm is related to tachycardia and higher pressure in the left atrium

In later stages of the disease some or all of these data may become permanent because an organic mitral lesion has been established

APEX CARDIOGRAM (LOW FREQUENCY TRACING)

The cardiogram may be useful in order to ascertain the phase of the additional diastolic sound and in the differentiation between loud third sound (ventricular gallop) and opening snap of the mitral valve (Fig 158) The latter has a more serious meaning because it indicates a well established lesion of the mitral valve and probably an initial mitral stenosis (p 329)

ELECTROKYMOGRAM

Electrokymographic tracings of the left atrium have been studied by the author and Magri [7] Some cases of rheumatic disease with a systolic murmur present a normal tracing others present evidence of increased pressure and initial regurgitation most of them however present a new plateau like pattern *indicating transmission of intraventricular pressure to the left atrium* (p 321) This does not imply valvular damage because acute carditis may also cause regurgitation However repeated examinations were found very useful in the evaluation of myocardial and valvular lesions

CONCLUSIONS

The graphic tracings are helpful in the diagnosis of rheumatic carditis Among them the most useful are

1 The electrocardiogram (changes of rate or rhythm prolongation of P R and Q T minor evolutionary changes of S T and T)
2 The phonocardiogram (systolic apical and pulmonic murmurs short diastolic rumble triple rhythm)
3 The electrokymogram (typical pattern over the left atrium)

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CHAPTER 41

Mitral Valve Lesions

MITRAL INSUFFICIENCY

Mitral insufficiency is usually the first result of a rheumatic lesion of the mitral valve Frequently later a partial fusion of the two leaflets causes some degree of stenosis However even in advanced cases mitral insufficiency may predominate to such an extent that the clinical picture is that of regurgitation only Although rheumatic fever is the most frequent cause, mitral insufficiency may be due to endocarditis from other causes, whenever the endocarditis of the mitral valve is caused by bacteria mitral insufficiency is the most important or the only consequence The graphic methods are of importance for the diagnosis

Electrocardiogram

Increased voltage of the ventricular complex in the limb and chest leads is not unusual Left axis deviation may be noted whenever the left ventricle is enlarged (Fig 145) Thus QRS has waves mainly directed upwards in aVL and in 1 downwards in aVF and 3 A qR complex is noted in the chest leads V4 to V6 If the left ventricle is markedly hypertrophied (as in most of the advanced cases) some degree of broadening of the complex is present inverted T in aVL 1 and in the chest leads V5 V6 may also occur The P wave is frequently tall notched or diphasic as a result of left atrial enlargement (Fig 222) The P R interval may be slightly prolonged reach

ing from 0 20 to 0 24 second as a result of an old rheumatic lesion of the conducting system Atrial fibrillation is frequent, especially if the patient is in failure, or has been digitalized for several years

Phonocardiogram

In initial or slight insufficiency a simple prolongation of the central phase of the first sound is present, later, the vibrations of the first sound continue during the first half of systole with decreasing intensity When mitral insufficiency is established, three types of murmurs can be recorded

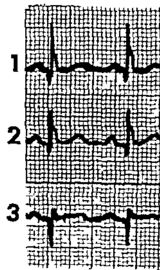


FIG 145 Electrocardiogram with left axis deviation (standard limb leads) Case of rheumatic heart disease with mitral insufficiency (no hypertension no aortic lesions)

1 A systolic murmur with gradually decreasing vibrations both high and low pitched (Fig 146A) This systolic murmur in decrescendo is the most common finding

2 A systolic murmur which continues throughout all systole with the same intensity, the vibrations are mostly high pitched (soft murmur) This all systolic murmur *is less common and is found* mostly in children (Fig 146 C)

3 A late systolic murmur with increasing intensity before the second sound This systolic murmur in crescendo is rare

The first high vibration of the first sound complex may be high (closing snap of the mitral valve, Fig 146 A B) The second high vibration of the second sound complex may become more visible and high pitched (opening snap of the mitral valve, Fig 147 B This phenomenon however is already evidence of a more severe damage to the valve and possibly of initial stenosis (Figs 153 and 154)

One of the diastolic sounds (third sound fourth sound) or both, may become high When only one of them is loud auscultation reveals a *triple rhythm* (Figs 146 A and B) When both are loud a diastolic rumble is usually heard (Figs 147 A D) An incorrect diagnosis of mitral stenosis may be made in such cases upon clinical auscultation This is more common in children [9] but is frequently observed also in adults [11]

Tracings recorded over the pulmonary artery may show a distinct systolic murmur and a loud or split second sound (Fig 147 B)

Apex Cardiogram

The low frequency tracing of the apex often shows the following changes [11] [9] quick rise followed by sudden drop before the end of the first

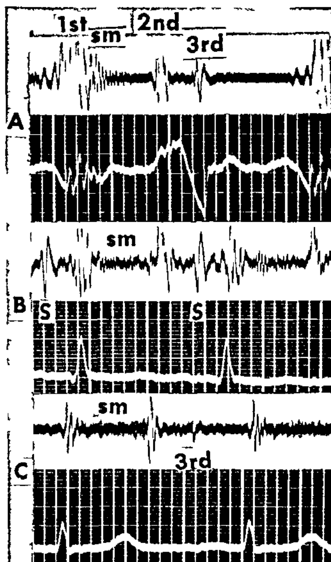


FIG 146 Cases of rheumatic heart disease with mitral insufficiency Stethoscopic phonocardiograms compared with a low frequency tracing of the apex in *A* with the ecg in *B* and *C*
A Systolic murmur in decrescendo at apex (*sm*) triple rhythm of ventricular type (*3rd*) *B* Faint systolic murmur at apex triple rhythm of summation type (*S*) *C* Loud high pitched all-systolic apical murmur in a child of 10 third sound

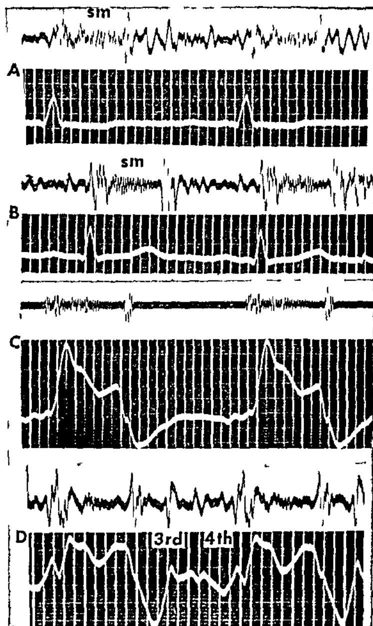


FIG 147 Cases of rheumatic heart disease with predominant mitral insufficiency

A Loud all systolic murmur at apex (*sm*) loud, low-pitched diastolic sounds Stethoscopic tracing and ecg *B* Systolic musical murmur, and split second sound over pul monic area Stethoscopic tracing and ecg *C*, Apical systolic murmur high apical thrust Logarithmic and low-frequency tracings at apex *D* Faint systolic murmur Loud diastolic sounds (quadruple rhythm) Stethoscopic and low frequency tracings at apex

sound complex low level of the point 2b, at times high waves during diastole (both waves 3 and 4 are high) (Fig 147 D)

Low frequency tracings recorded outside the apex (regional cardiograms) show

1 High atrial wave (4) over the midprecordium (strong left atrial contraction)

2 High vascular wave (p) at the 2nd left interspace (high pulmonic pulsation)

Jugular Vein Tracing Pneumocardiogram

Both these tracings often show large waves

Electrokymogram

This tracing is of primary importance for diagnosis

The electrokymogram should be recorded as a border tracing of the left atrial appendage and of the posterior border of the left atrium in both oblique positions (p 183) A densogram of the left atrium can also be recorded in certain cases as well as a border tracing of the left atrial appendage

As already described (p 191) the normal tracings *of the left atrium* consist of a sharp negative wave in presystole (inward motion caused by atrial contraction) and a more rounded negative wave in systole (inward motion caused by lowering of the a v septum only partly compensated by venous flow into the atrium) In cases of mitral insufficiency a new pattern is visible

1 The presystolic negative wave is deeper and broader (unless there is atrial fibrillation) on account of stronger atrial contraction (Fig 149 A) *

2 The systolic negative wave has disappeared and is substituted by a *positive plateau* There is a rapid rise in early systole a sharp angle a straight or concave line in systole another sharp angle then a rapid drop after the opening of the mitral valve (Figs 148 149)

The systolic plateau is the graphic expression of a systolic swelling of the left atrium during ventricular systole caused by the blood which regurgitates from the left ventricle It resembles a tracing of left atrial pressure recorded in animals with experimental mitral insufficiency[9] or in patients with early rheumatic disease[10] It is similar to a tracing of intraventricular pressure (Fig 79)

Graphic differentiation between this plateau and possible arterial pulses is usually not difficult (Fig 150) The various stages of insufficiency from the slightest degree to the severe regurgitation are revealed by successive substitution of the plateau for the normal systolic trough (Fig 151) It may happen that one of the oblique positions reveals the typical pattern while

* This presystolic wave is especially deep over the left atrial appendage this fact is partly due to the strong contraction of this structure which causes important motion phenomena

the other does not It is likely, though not proven that this indicates a moderate regurgitation The existence of this diagnostic pattern which had been previously detected by roentgenkymography,[7, 4] was demonstrated by the author with Fleischner[1, 1] and confirmed by various other researchers[5 9 19 ,5 8]

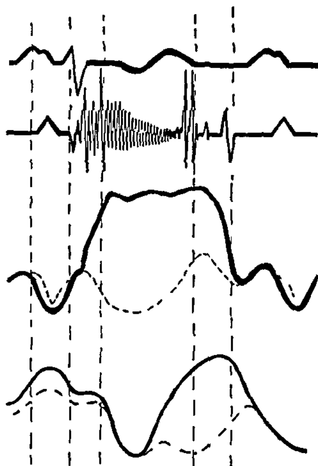


FIG 148 Scheme of the typical changes in mitral regurgitation Electrocardiogram phonocardiogram electrokymogram of left atrium electrokymogram of pulmonary veins pulmonary capillary' pressure Dotted lines indicate normal tracings

The electrokymogram of the *pulmonary veins* presents two typical changes

1 A high and broad positive wave in presystole, equivalent to the a wave of the jugular tracing

2 A high positive plateau in systole, the result of increased pressure and transmission of the plateau like tracing of pressure from the left atrial cavity It reveals the fact that during systole the blood cannot flow from the pulmonary veins into the left atrium[13]

Esophagocardiogram

This tracing recorded at the level of the left atrium shows a typical pattern It consists of a high and peaked positive wave instead of the negative wave which is normally present during ventricular systole[23, 6] The importance of this tracing is decreased by the ease of recording an electrokymogram, and the typical pattern revealed by that method However an esophagocardiogram can be studied if one lacks an electrokymograph and is more accurate if recorded by an electromanometer (piezo esophagocardiography[35])

Conclusions

The graphic signs of mitral insufficiency having diagnostic value are

1 Evidence of a systolic murmur at the apex (phonocardiogram)

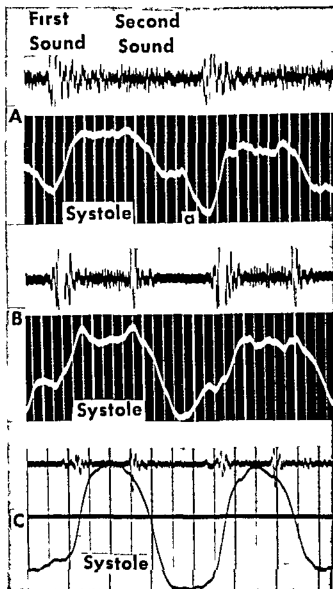


FIG 149 Rheumatic heart disease Mitral insufficiency Electrokymograms of the left atrium revealing a plateau like rise during ventricular systole *A* patient with sinus rhythm *B* patient with atrial fibrillation *C* patient with atrial fibrillation and extremely severe regurgitation *a* atrial wave

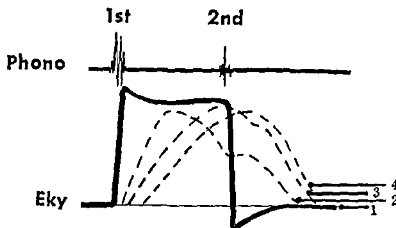


FIG 150 Electrokymogram of left atrium Sketch showing differentiation of the *positive plateau 1*, from the pulse of the pulmonary artery *2*, from that of the hilar vessels *3*, or that of the lungs *4* Shape and relationship of the waves to the heart sounds are different (From Luisada and Fleischner, courtesy of *Am J Med*)

2 Evidence of an increase in volume of the left atrium during ventricular systole (electrokymogram of the left atrium esophagocardiogram)

RELATIVE MITRAL INSUFFICIENCY

In general the electrocardiogram is normal or reflects changes caused by coronary or hypertensive heart disease or by lesions of other valves

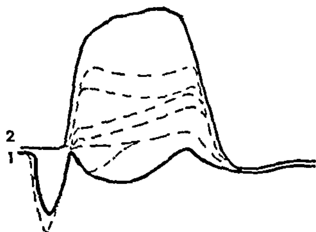


FIG 151 Scheme of electrokymograms of left atrium in mitral lesions Line *1*, normal, Line *2*, severe regurgitation and atrial fibrillation Stippled lines represent intermediate stages

The phonocardiogram presents a series of high pitched vibrations of poor amplitude during systole There may be no real systolic murmur while the first sound is prolonged There may be accentuation of those low pitched vibrations caused by vascular distention which are frequently found in normal subjects and represent the last phase of the first sound (p 44)

The electrokymogram of the left atrium may be entirely normal However, if

dilatation of the mitral ring causes an actual and important regurgitation of blood, the typical pattern of regurgitation (plateau) may be present (p 321)

MITRAL STENOSIS

Narrowing of the mitral valve (mitral stenosis) is usually one of the late results of rheumatic endocarditis and is nearly always accompanied by regurgitation However in certain cases whether through a special type of deformity of the valves or on account of lack of growth of the ostium (mild

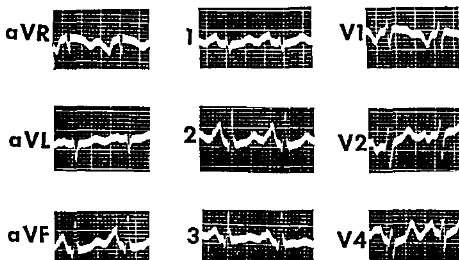


Fig 152 Electrocardiogram of a case of mitral stenosis with sinus rhythm Right axis deviation Right ventricular hypertrophy Typical P wave

endocarditis in the early stages of life) mitral stenosis though severe seems clinically unaccompanied by regurgitation

Electrocardiogram

Several changes may be observed (Fig 152)

1 High diphasic or notched P wave in the limb leads Diphasic or inverted P in V1 and V2

2 A prolonged P R interval (0 21–0 24 second)

3 Right axis deviation revealed by comparing aVL with aVF, or lead 1 with lead 3

4 A shift of the transitional zone in the chest leads revealing that the right ventricle forms all of the anterior portion of the heart and even the apex (rS complex in V1 to V5)

5 Possible right bundle branch block or intraventricular block

6 Possible disturbances of the rate and rhythm consisting of atrial premature contractions atrial flutter or fibrillation, paroxysmal atrial tachycardia or ventricular tachycardia

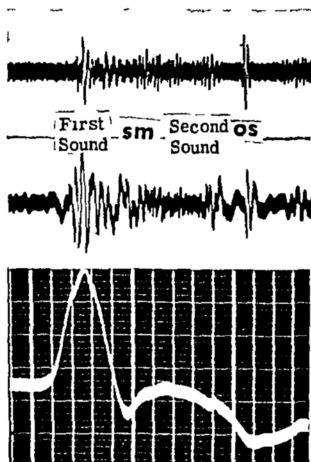


Fig 153 Mitral stenosis Opening snap of the mitral valve (*os*) *Above* logarithmic tracing *Center* stethoscopic tracing *Below* linear tracing (low frequency tracing—or apex cardiogram)

Phonocardiogram

The findings vary with different auscultatory signs and over different areas

1 There may be a *presystolic murmur in crescendo* at the apex This reveals itself by a series of oscillations in late diastole which increase in intensity and then fuse with the 1st sound (Fig 155 B and C) If the conduction time is long the presystolic murmur may be separated from the first sound and loses its crescendo type In extreme prolongation of conduction this murmur may even fall in early diastole (Fig 156) This murmur disappears if there is atrial fibrillation

2 There may be a *rumbling diastolic murmur* also at the apex This is revealed by a series of irregular and low pitched vibrations (Fig 155) They start some time after the main oscillation of the second sound-complex with a small but rapid vibration (the opening snap of the mitral valve) they continue until middiastole or late diastole with decreasing intensity, and then disappear There may be a pause between this murmur and the presystolic murmur if diastole is long If, on the contrary, diastole is short, the diastolic murmur continues and merges with the presystolic murmur A high vibration equivalent to a gallop sound may be recognized within the murmur if the stenosis is not too severe

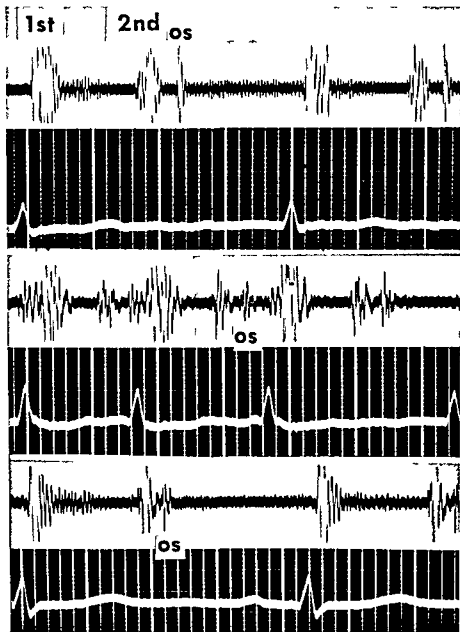


Fig 154 Rheumatic heart disease Mitral stenosis Opening snap of the mitral valve (*os*) in three different cases The upper phonocardiogram is logarithmic the others are stethoscopic tracings

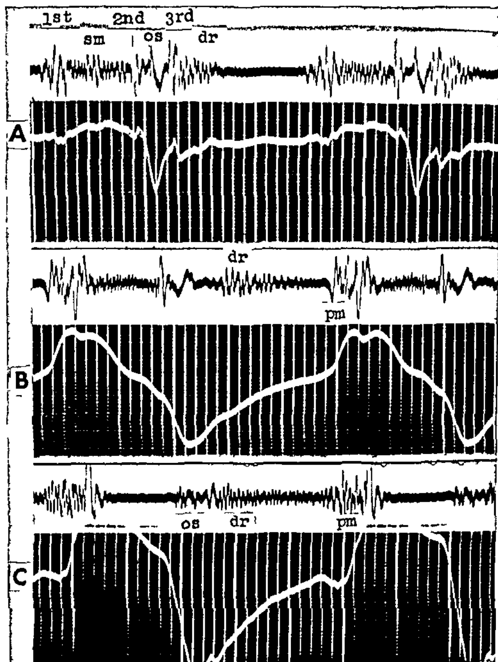


Fig 155 Rheumatic heart disease Mitral stenosis Stethoscopic phonocardiograms recorded within the apex in three different cases compared with linear tracings (low frequency tracings)

A, Systolic murmur, opening snap (*os*) loud third sound, early-diastolic murmur (*dr*)

B Presystolic (*pm*) and systolic murmurs, diastolic rumble (*dr*) Slow filling of left ventricle

C, Typical presystolic (*pm*) murmur no systolic murmur opening snap (*os*) third sound long diastolic rumble (*dr*) Slow filling of left ventricle

3 The *opening snap of the mitral valve* (Figs 153 and 154) is a typical vibration which alone is sufficient to diagnose a lesion of the mitral valve and probably mitral stenosis It is a rapid vibration which is separated from the main vibration of the second sound complex by an interval of from 0 08 to 0 12 second [17, 80] This interval is of variable length from cycle to cycle if there is atrial fibrillation [80-81]* The snap is frequently followed by diastolic vibrations but it may be isolated It is recorded best at the midprecordium It should be distinguished both from the physiologic opening sound of the mitral valve and from the third sound (or gallop sound) (Fig 158) The

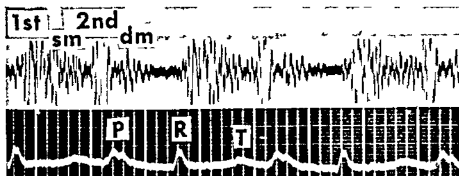


FIG 156 Rheumatic heart disease Mitral stenosis Long P R interval (0 26) Phonocardiogram and electrocardiogram The murmur caused by the atrial contraction (*dm*) takes place in early diastole and is distant from the first sound

opening snap is an accentuation of the physiologic opening sound but has a higher pitch and takes place later on account of delayed opening of the mitral valve According to Holldach s observations [8] the opening snap takes place in those cases of mitral stenosis where the leaflets are still flexible not in those with callous cusps It coincides with the point 2b of the cardiogram (Figs 153 and 158) with the point *t* of the pneumocardiogram and with the *v* wave of the jugular tracing (if the latter is not delayed) It may be recorded over a large area of the chest at times even outside the precordium

4 It is common to observe the vibrations of a *systolic murmur at the pulmonic area* Curiously enough these vibrations are frequently louder in the early stages of the disease unless severe dilatation of the pulmonary artery is present There frequently is a *split second sound* (Fig 157) The distance between the two phases of this split sound varies from 0 04 to 0 08 second Very seldom is the splitting audible when the interval is below 0 06

5 There may be an *early diastolic murmur in decrescendo* at the pulmonic

* According to Kuo and Schnabel (Am Heart Assn 1952) both the delay between QRS and first sound and that between main vibration of second sound and opening snap remain fixed whenever there is severe mitral or aortic insufficiency

area due to pulmonic insufficiency (*Graham Steell murmur*) This murmur is usually of short duration, appearing only during congestive failure (Fig 144 C) A similar, persistent murmur indicates sclerotic or endocarditic pulmonic insufficiency [18] An aortic diastolic murmur in this region is, of course extremely common

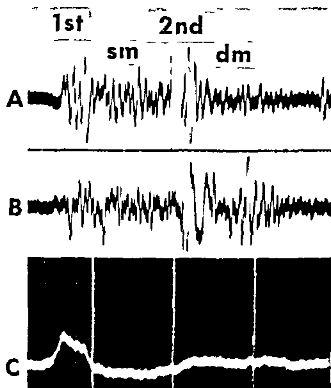


FIG 157 Rheumatic heart disease Mitral stenosis and insufficiency
A Stethoscopic phonocardiogram over pulmonic area Loud systolic murmur (*sm*) split second sound
B Stethoscopic phonocardiogram at apex Fainter systolic murmur, opening snap of the mitral valve, loud diastolic murmur (*dm*)
C Electrocardiogram

When atrial fibrillation is present the interval between QRS of the electrocardiogram and the main vibration of the 1st sound complex may vary becoming longer when the preceding diastole is short on account of delayed mitral valve closure Thus the interval between QRS and the first high vibration of the first sound may become variable [4, 10, 1*]

* According to Kuo and Schnabel (Am Heart Assn 1952) both the delay between QRS and first sound and that between main vibration of second sound and opening snap remain fixed whenever there is severe mitral or aortic insufficiency

When a case of mitral stenosis has no audible murmur either no murmur is recorded (rare) or there are low pitched inaudible vibrations during diastole (subsonic murmur) In both cases the murmur may appear or become audible after exercise

Cardiogram

The low frequency tracing of the apex (apex cardiogram) may reveal the following phenomena (Fig 155)

1 Slow rise of the tracing during the tension period with a delay of the peak 1a (delayed closure of the mitral valve) [4, 8]

2 Slow descent of the tracing after the point 2a (delayed opening of the mitral valve) and a slow rise afterwards (slow filling in early diastole) [28]

3 Small or absent atrial wave (difficult filling of the left ventricle)

The low frequency tracing of the second left interspace (regional cardiogram) often reveals a high systolic wave (large pulsation of the pulmonary artery) That recorded over the third left interspace may present a high atrial wave (large pulsation of the left atrium)

Epigastric Tracing

The epigastric tracing frequently shows a high atrial wave (large pulsation of the right atrium) and a high systolic pulsation (strong contraction of the right ventricle) [31]

This tracing frequently shows a diphasic H wave a high L and a deep M [33]

Electrokymogram

The electrokymogram of the left atrium studied by the author with Fleischner [6] frequently reveals the typical pattern of mitral regurgitation (p 321) even in cases where no systolic murmur is heard or recorded This confirms that the occurrence of pure mitral stenosis is less common than previously thought The stenosis of the mitral valve is frequently revealed by a small notched or prolonged atrial wave in presystole [20] If there is atrial fibrillation the atrial wave disappears Similar findings were reported by others in subsequent studies [5, 9, 19, 5] Andersson[31] observed increased depth of the presystolic wave over the left atrial appendage not over the left atrial border It is likely that the increased depth is due not to a change in volume but to a positional change on account of the high mobility of the hypertrophied appendage

Cardiomanometry

Catheterization of the right heart reveals that the pressure of the right ventricle pulmonary artery and pulmonary capillaries is frequently increased This is due to the narrowing of the mitral valve plus structural changes of

the arterioles, and frequently also pulmonary vasoconstriction Average values for the pulmonary artery were 30/16 in patients without heart failure and 69/34 in patients with failure[2, 3] These values should be compared with the normal average figures of 20/9 Direct recording of pressure in the left atrium before commissurotomy[7] has revealed pressures of 25–30 mm Hg during ventricular systole, caused by the insufficiency of the mitral valve Eval

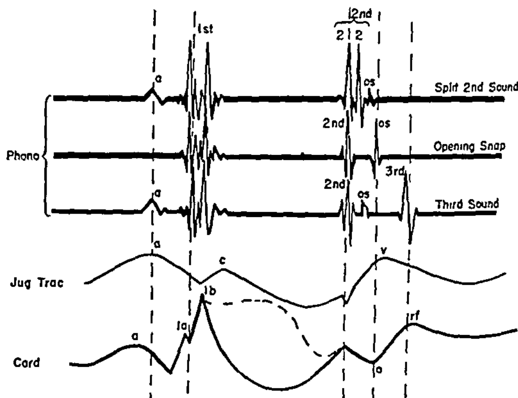


Fig 158 Differential diagnosis between opening snap of the mitral valve third heart sound and split second sound

uation of the mitral area and of the regurgitant flow has been attempted on the basis of data supplied by cardiomanometry[6] The study of the right ventricular, pulmonic, and pulmonic capillary pressures at rest and during exercise has been used for evaluation of arteriosclerosis of the pulmonary vessels Even though this evaluation is still not exact the data are of interest *Lack of increase of pulmonic capillary pressure during exercise while right ventricular systolic pressure is very high and diastolic pressure is normal has been accepted as evidence of arteriosclerotic narrowing of the pulmonary vessels*

Conclusions

The most important graphic signs of mitral stenosis having diagnostic value are

1 Changes of the electrocardiogram
2 Existence of typical murmurs (phonocardiogram)
3 Evidence of increased volume of the left atrium during ventricular systole due to associated mitral insufficiency (electrokymogram of the left atrium)

RELATIVE MITRAL STENOSIS

It has been proven that whenever the left ventricle is severely dilated the blood penetrating through the mitral valve creates whirlpools which cause a rumbling diastolic murmur This murmur may be recorded by phonocardiography in cases of acute rheumatic fever coronary heart disease or aortic insufficiency It can be often differentiated from that of absolute mitral stenosis because composed of high and irregular vibrations which start rather late in diastole and are not preceded by an opening snap of the mitral valve (Figs 161 162 and 163) These vibrations may be recorded in certain cases over a wide surface of the precordium [14]

It should be kept in mind that upon auscultation the presystolic murmur of mitral stenosis may be simulated by a crescendo type of the first sound [1 11 16] and by triple rhythms or other abnormal cadences [14 16]

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CHAPTER 42

Aortic Valve Lesions

AORTIC INSUFFICIENCY

Similar graphic findings are obtained in the various forms, the syphilitic the bacterial and the rheumatic The only important difference is that the syphilitic form is usually associated with 'relative stenosis while the others are frequently associated with absolute stenosis (p 346)

Electrocardiograms

The most typical finding is left axis deviation, revealed by comparison of aVL with aVF and lead 1 with lead 3 (Fig 159) Additional changes may consist of

1 Prolongation of the P R interval

2 Evidence of left ventricular hypertrophy and anterolateral ischemia (Fig 220) The former consists of increased voltage, a slight prolongation of QRS (not exceeding 0 11 second) and a delay of R over the beginning of the complex of more than 0 04 in V5–V6 The latter is revealed by lowering or inversion of T in aVL (and lead 1) and in the chest leads V4 to V6

3 Left intraventricular block or bundle branch block

4 Abnormalities of S T and T other than those related to ischemia or bundle branch block

These changes are connected with either rheumatic lesions (*P R*) or

coronary disease The latter is favored by the hemodynamic changes caused by the valvular lesion and may be present in the syphilitic form

5 Left ventricular premature beats are common Ventricular tachycardia may occur

Phonocardiogram

Tracings recorded over the aortic area show that the main vibration of the second sound complex is followed by a series of other vibrations of high pitch They are usually of limited amplitude in comparison with the auditory impression and decrease gradually (Figs 160, 164 and 165) When a broken or everted valve is the cause of regurgitation the vibrations are higher and may be regular (*musical murmur*) (Fig 160 C)

The characteristics of the *diastolic murmur* vary In some cases the vibrations follow each other with a decrescendo type In other cases the vibrations have a crescendo decrescendo type as described by Sprague *et al*[14] The murmur sometimes ends in early diastole while in most cases continues with small vibrations throughout all diastole If the murmur is musical as in cases with an everted aortic valve or calcification of one of the leaflets the vibrations are regular and present a concertina like appearance

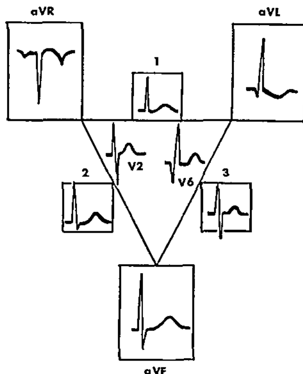


FIG 159 Electrocardiogram in aortic insufficiency (Sketch from actual case)

The first sound may be impure (low or prolonged) because of myocardial damage There may be a high vibration (*aortic opening click*) which slightly precedes[15] or coincides[3] with the rise of the carotid pulse If it precedes this rise it is an opening click of the semilunar valves if it coincides with it it is due to the systolic distention of the dilated aorta

Vibrations due to a *systolic murmur* are common (Figs 160 and 161) they

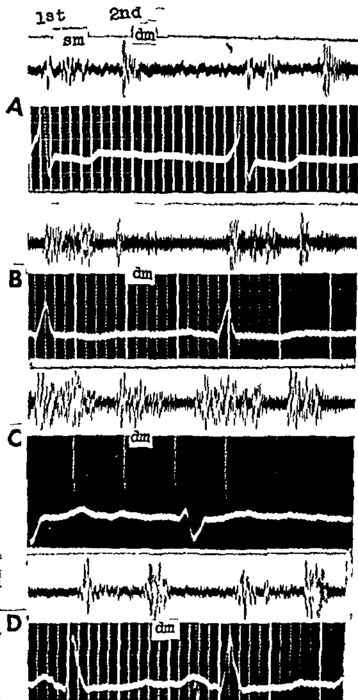


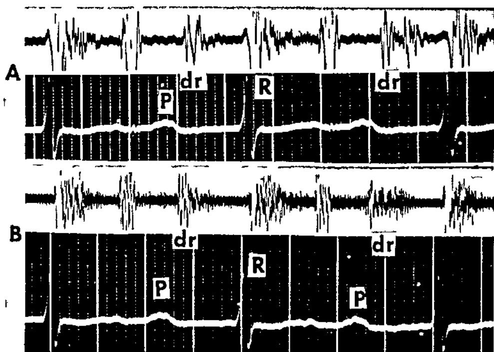
FIG 160 Luetic heart disease Aortic insufficiency Phonocardiograms recorded over the aortic area in four different cases Case *C* had an everted aortic valve with sudden appearance of the murmur following strenuous exertion *dm* soft early-diastolic murmur

are sımılar to those encountered ın organıc aortıc stenosıs relatıve stenosıs of the aorta and aortıtıs

Studıes of the author on the *Austın Flınt murmur*[11] have revealed the followıng

1 In some cases a trıple rhythm ıs present

2 In others the low amplıtude of the first phase of the first sound and the hıgh amplıtude of the openıng clıck gıve the ımpressıon of a presystolıc



Fɪɢ 161 Aortıc ınsufficıency coronary heart dısease hyperthyroıdısm *A* stethoscopıc phonocardıogram at apex *B* logarıthmıc phonocardıogram at apex There ıs a mıd dıastolıc rumble (*dr*) connected wıth the atrıal contractıon which becomes occasıonally splıt (*dr dr*) Subsequent tracıngs revealed a trıple rhythm There also ıs a systolıc murmur (unmarked)

murmur to the unaıded ear A delay ın the occurrence of the first sound[3] may contrıbute to thıs

3 In others slow systolıc vıbratıons arısıng ın the aorta are louder than the first sound and may be easıly mıstaken for ıt (Fıg 163 A)

Therefore ın most cases eıther there ıs a trıple rhythm or the presystolıc murmur ıs due to an auscultatory ıllusıon

Subsequent studıes of the author wıth Montes[12] have shown that ın rare cases the phonocardıogram reveals a dıastolıc rumble whıch may be mıstaken

for that of mitral stenosis (Figs 161, 162 and 163) However, the following differential data may be observed in such cases

1 There is no opening snap of the mitral valve
2 The murmur starts rather late in diastole
3 The vibrations are of unusually large amplitude
4 They are recorded over a large area, sometimes over the entire precordium

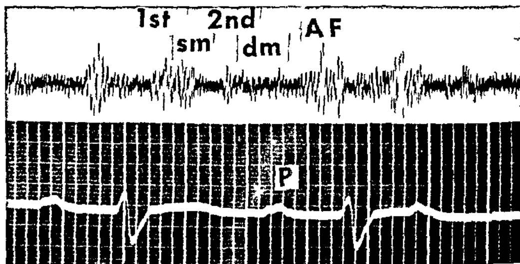


FIG 162 Luetic and coronary heart disease Aortic insufficiency Austin Flint murmur (*AF*) (relative mitral stenosis) Phonocardiogram over midprecordium A diastolic rumble is present during atrial contraction Subsequent tracings failed to reveal this rumble

Phonoarter ograms and Blood Pressure

Arterial sound vibrations may present the following types [10]

1 There may be a single, extremely loud, sound during the expansion of the artery (*pistol shot sound*)

2 There are two sounds The first of these falls during the expansion of the artery while the second falls during arterial collapse and precedes the dicrotic wave (*double tone of Traube* [Fig 164 A])

3 There are two murmurs These replace the two sounds and are favored by compression below the point of recording They are more easily observed over the femoral artery (*double murmur of Duroziez* [Fig 164B])

Arterial pressure recorded by the graphic method (p 154) has usually a high systolic and a low diastolic level However the latter is seldom as low as indicated by clinical measurements

Cardiogram

In most cases the low frequency tracing of the apex (apex cardiogram) shows a high early systolic wave followed by a deep depression In certain cases however, a high systolic plateau is present (Fig 165) If the record is taken outside the apex a deep systolic depression represents the main aspect of the tracing

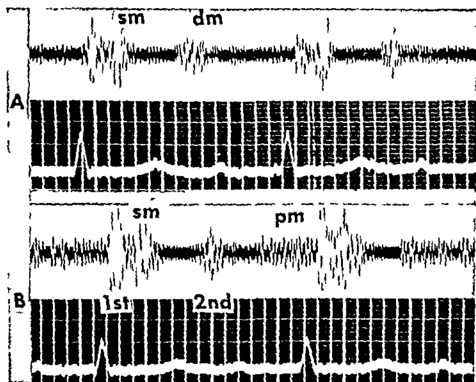


FIG 163 Luetic and coronary heart disease Aortic insufficiency Austin Flint murmur (relative mitral stenosis) confirmed by autopsy *A* Logarithmic tracing at the base *sm* systolic murmur *dm* soft diastolic murmur *B* Logarithmic tracing at the apex *sm* systolic murmur *pm* presystolic murmur in crescendo

The atrial wave is often high in the syphilitic and arteriosclerotic forms Rapid filling is often accompanied by a high wave in early diastole which if marked is typical (the blood regurgitating from the aorta increases the rapidity of filling)

Tracings recorded either over the second right interspace or the suprasternal notch (aortograms p 70) reveal a high systolic wave

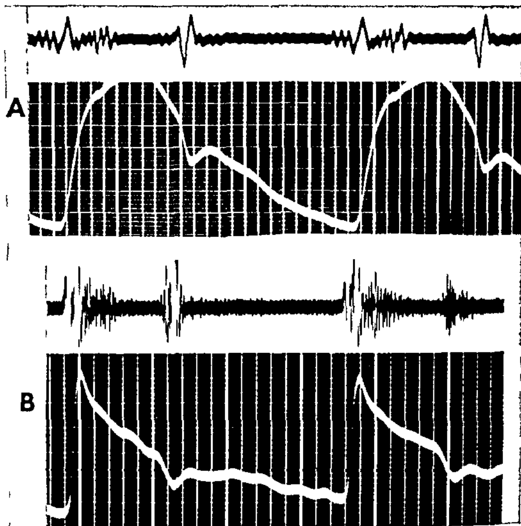


FIG 164 Two cases of aortic insufficiency Femoral sounds and pulses *A* Double tone of Traube *B* Double murmur of Duroziez

Pulse Tracings

The typical pulse curve presents a rapid ascent a quick descent and a small dicrotic wave (Fig 166) The quick descent is systolic and therefore not directly connected with the regurgitation of blood The comparison of various tracings at different sites shows that transmission of the pulse wave is rapid in all arteries

Jugular Vein Tracing

The only remarkable finding is a deep systolic collapse

Pneumocardiogram

The systolic waves are very deep There may be only a deep $v1$ or a fusion of $v1$ with $v2$ The point s is high and the point t is even higher (Fig 167) This is due to the regurgitation of blood into the left ventricle which rapidly increases the content of blood of the chest

Roentgenkymogram

The dilatation of the aorta occurs very early but the pulse wave moves slowly at first

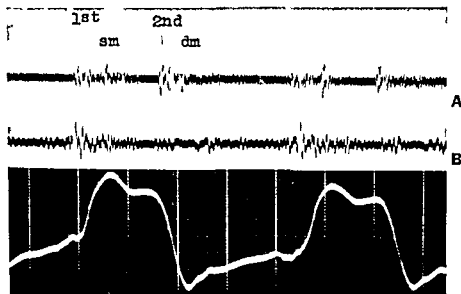


FIG 165 Rheumatic heart disease mitral stenosis and aortic insufficiency Logarithmic phonocardiogram over aortic area *A* Stethoscopic phonocardiogram at apex *B* Low frequency tracing at apex (apex cardiogram)

Electrokymogram

The border tracing of the aortic arch reveals a rapidly expanding and rapidly collapsing pulse (Fig 166) the absolute magnitude of the pulse is increased [8] The border tracing of the left ventricle shows a pulsation made of a rapid and large drop (rapid ventricular contraction) and a rise in early diastole which is faster than in normal subjects[9] (Fig 168) The latter is due to the fact that the left ventricle is distended by two streams the normal left atrial blood and the aortic regurgitant blood

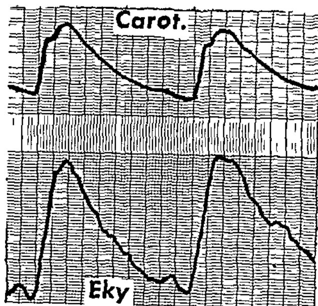


Fig 166 Aortic insufficiency Carotid tracing and electrokymogram of aortic arch (border tracing) Rapid rise and rapid collapse of the pulses

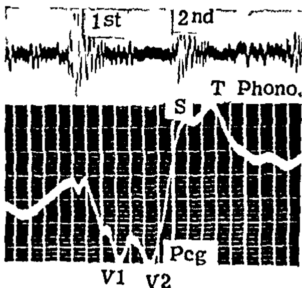


Fig 167 Pneumocardiogram in aortic insufficiency High S T tract due to the regurgitation Deep V1-V2 due to large ejection

Conclusions

The graphic signs having diagnostic importance are (Fig 169)

1 The electrocardiographic changes
2 The soft high pitched early diastolic murmur (phonocardiogram)
3 The rapidly expanding and rapidly collapsing arterial pulse (carotid tracing electrokymogram of the aortic arch)
4 The unusual sound or murmur during the collapse of the peripheral arteries (phonoarteriogram)
5 The increased rapidity of filling of the left ventricle (electrokymogram of left ventricle apex cardiogram)

RELATIVE AORTIC INSUFFICIENCY

Functional insufficiency of the aortic valve is due to incomplete closure caused by weakness of the musculature of the left ventricle This has been described in heart failure in acute rheumatic disease and in the thyrotoxic heart [13] The possibility of a relative aortic insufficiency in severe hypertension has been also demonstrated [7, 8]

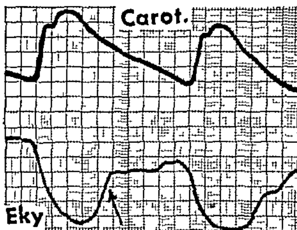


FIG 168 Aortic insufficiency Carotid tracing and electrokymogram of the left ventricle The rapid rise (arrow) in early diastole reveals the regurgitation of blood into the left ventricle

FIG 169 Scheme of the changes of graphic tracings in aortic insufficiency The dotted lines indicate the normal patterns, the full lines the abnormal

In these cases, the only graphic findings are the vibrations of the diastolic murmur in the phonocardiogram The murmur usually consists of three to five vibrations in decrescendo immediately after the second sound The pulse and blood pressure tracings, the electrocardiogram, and the electro kymogram fail to show typical data

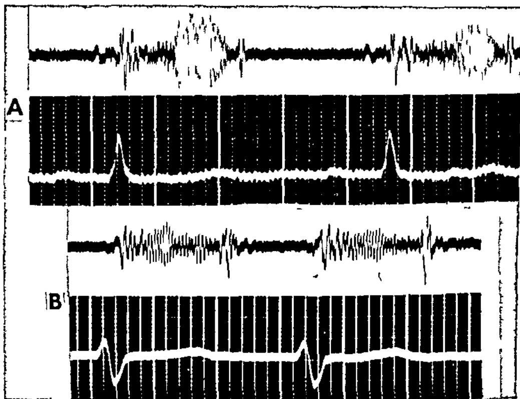


Fig 170 Two cases of calcific aortic stenosis Musical systolic murmurs over the aortic area Phonocardiograms and electrocardiograms In *A* the murmur is of a much higher pitch than in *B*

AORTIC STENOSIS

Aortic stenosis may be congenital it may be caused by rheumatic or bacterial endocarditis or by fibrosis or calcification of the valve (calcific aortic stenosis) often due to atherosclerosis In all these forms the graphic findings are directly related to the valvular lesion Rheumatic endocarditis frequently causes also mitral valve lesions and many of the above cases also have an aortic insufficiency

Electrocardiogram

The data are similar to those caused by aortic insufficiency (p 336) because they are related to the enlargement of the left ventricle and to the

possible association of coronary and myocardial lesions Sinus bradycardia is frequently observed Even during heart failure there may be a relative bradycardia

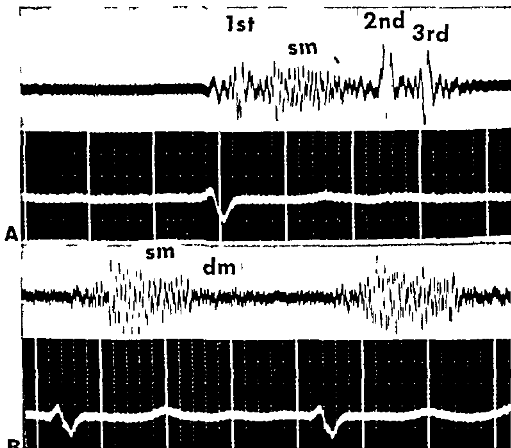


FIG 171 Rheumatic heart disease aortic insufficiency and stenosis Phonocardiograms (stethoscopic tracings)
A Apex *transmitted systolic murmur (sm) loud second and third sounds early* diastolic low pitched murmur (*dm*) *B* Aortic area diamond shaped systolic murmur (*sm*) absent second sound early-diastolic high pitched murmur (*dm*)

Phonocardiogram

The phonocardiogram gives evidence of the systolic murmur which is revealed by a series of vibrations of both high and low pitches The vibrations increase gradually and often assume the shape of a wave having maximum intensity in the second half of systole (*diamond shaped murmur*) (Figs 170 171 172 and 173) The second sound has a low amplitude and may disappear In certain cases the second sound is loud over the second right interspace (transmission from pulmonic area) but disappears over the first A loud fourth (atrial) sound is often present

Cardiogram

The ascending part of the systolic impulse is either slow or double (*camel back or staggered profile*) The systolic murmur may be accompanied by a series of vibrations which are the graphic expression of the thrill (Fig 172)

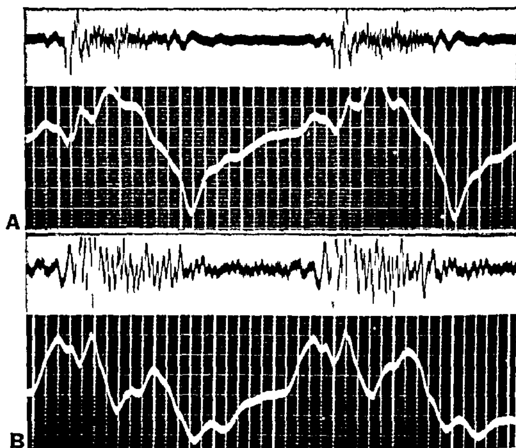


FIG 172 Calcific aortic stenosis Stethoscopic and linear tracings at the apex (*A*) and over epigastrium (*B*) rough systolic murmur staggered ventricular contraction

Arterial Pulse

The pulse tracing shows an ascending phase which is slower than normal As this phase is often interrupted by a well marked depression (*anacrotic notch*), the pulse is called anacrotic pulse If recorded at the suprasternal notch or on the carotid arteries the pulse tracing may show a series of vibrations which are simultaneous with the murmur (so called *carotid shudder*)[8] (Figs 173, and 174)

A

B

FIG 173 Two cases of calcific aortic stenosis Sound tracings and aortic pulses at suprasternal notch Slow rise in *A* anacrotic pulsation in *B*

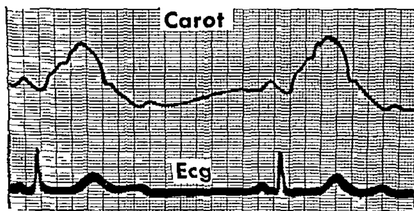


FIG 174 Carotid tracing and electrocardiogram in a case of aortic stenosis Anacrotic pulse

Blood Pressure

Graphic tracings of blood pressure frequently reveal an *auscultatory gap*. This consists of the absence of arterial sounds in a zone between systolic and diastolic pressures. While the sound tracing reveals the "gap" the pulse tracing does not. The phenomenon seems to be intimately connected with the anacrotic depression of the pulse and the less rapid distention of the artery during that phase of the cycle (Fig 175)[1, 6]

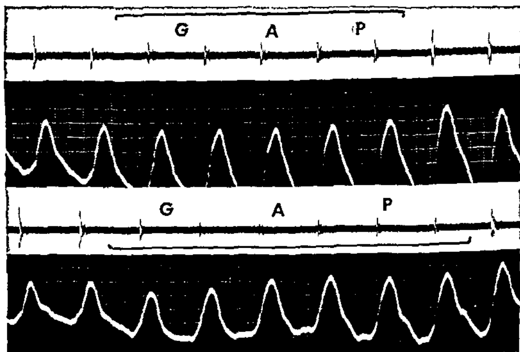


FIG 175 The auscultatory gap in a case of aortic stenosis (two successive measurements)

Electrokymogram

The border tracing of the aortic knob presents an anacrotic depression similar to that exhibited by the carotid pulse, the border tracing of the left ventricle reveals abnormalities of contraction similar to those exhibited by the apex cardiogram

Conclusions

The graphic signs of diagnostic importance are (Fig 176)

1 Electrocardiographic data

2 Existence of a diamond shaped murmur and of a weak second sound over the aortic area (phonocardiogram)

3 Proof of an obstruction located at the aortic valve (apex cardiogram aortogram carotid tracing electrokymogram of the left ventricular border and the aortic arch)

4 Existence of an "auscultatory gap in the record of the Korotkow sounds and of an anacrotic depression in the tracing of the arterial pulse

RELATIVE AORTIC STENOSIS

Patients with a dilated ascending aorta usually present a systolic murmur caused by the disproportion between normal valvular opening and large

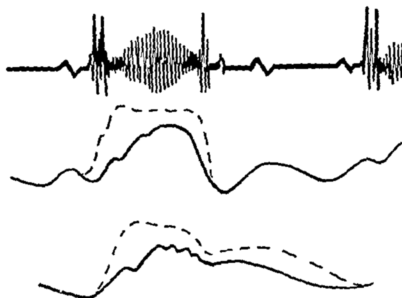


FIG 176 Scheme of the changes in aortic stenosis The dotted lines indicate the normal patterns the full lines the abnormal

arterial vessel Patients with atherosclerosis and fibrosis of the aortic leaflets also may present a systolic murmur over the aortic area Both groups of patients might be diagnosed as having aortic stenosis while no obstacle to the outflow from the left ventricle is present The phonocardiogram cannot be used for the differential diagnosis of these cases because the murmur is diamond shaped and presents irregular vibrations as in cases with absolute stenosis It should be noted however that in relative stenosis the vibrations are less high and the second aortic sound is normal or loud In cases with organic stenosis the apex cardiogram the aortogram of the suprasternal notch and the carotid tracing reveal typical data which are evidence of obstruction they fail to do so in cases with relative stenosis or valvular fibrosis Similar data are supplied by the electrokymogram

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CHAPTER 43

Tricuspid Valve Lesions

TRICUSPID INSUFFICIENCY

Tricuspid insufficiency is extremely rare as a pure defect It is much more frequent in combination with tricuspid stenosis, mitral defects, or an atrial septal defect

Electrocardiogram

There is a right axis deviation Atrial fibrillation is common and occurs early (Fig 177) Ventricular premature beats and attacks of ventricular tachycardia are frequent in the later stages A peaked and diphasic P wave in lead V1[1] is frequent in the early stages and reveals right atrial hypertrophy (Fig 222)

Phonocardiogram

This tracing records a systolic murmur in decrescendo or a continuous systolic murmur over the tricuspid area and at the right of the sternum This murmur is frequently high pitched and becomes louder in inspiration[13] (Fig 178) Thus a comparison between tracings taken in inspiration and expiration permits differentiation from a mitral systolic murmur because the latter decreases or remains unchanged in inspiration Moreover this murmur is louder over the tricuspid area than at the apex a comparison between the apical and the tricuspid phonocardiogram excludes a transmitted murmur (Fig 179)

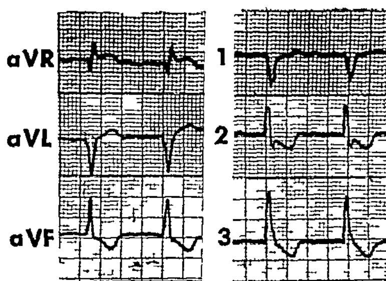


FIG 177 Electrocardiogram in a case of rheumatic heart lesion of the mitral and tricuspid valves Atrial fibrillation

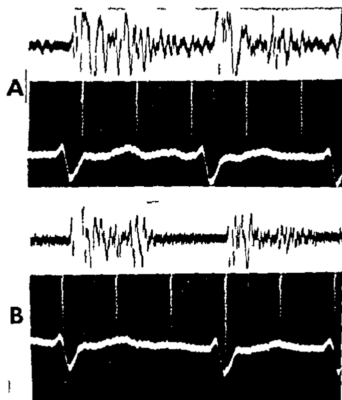


FIG 178 Rheumatic heart disease with mitral and tricuspid insufficiency and stenosis The systolic and diastolic murmurs recorded *over the tricuspid area* are louder in inspiratory apnea (*A*) than in expiratory apnea (*B*) indicating their connection with right heart pressure changes

Cardiogram

This tracing reveals that the region of the apex has a retraction or backward thrust during ventricular systole On the other hand there is a systolic forward thrust of the epigastrium the lower part of the sternum and the right precordial area [18] This double motion creates a see saw movement

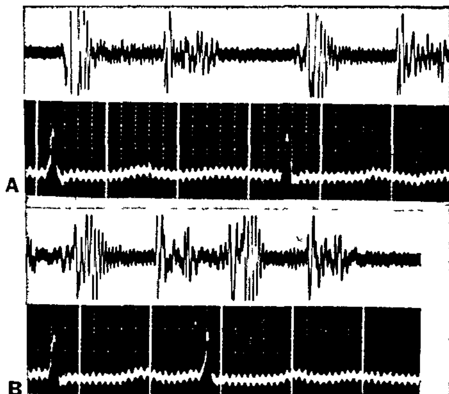


FIG 179 Rheumatic heart disease Mitral and tricuspid insufficiency and stenosis *A* Phonocardiogram over tricuspid area *B* Phonocardiogram at apex Different duration and loudness of the murmurs

Epigastric Tracing

This tracing may reveal a high early systolic wave (thrust of the right ventricle) followed by a late systolic pulsation (hepatic pulsation) However frequently the enlarged liver occupies the epigastrium and a pure hepatic tracing is recorded If the sinus rhythm is preserved there is a high atrial wave

Jugular Tracing Hepatic Tracing

Studies of these tracings have been made by MacKenzie[10] and in more recent years by Kerr and Warren[6] White and Cooke[17] Hallock and Clarke[5] Groedel[4] the author[9] and Sprague and co workers[11] Both tracings show a

high positive presystolic wave (unless atrial fibrillation is present), and a high positive, systolic wave The latter assumes in severe lesions the aspect of a positive, *systolic plateau* similar to a tracing of intraventricular pressure (Figs 180 and 181) * If the regurgitation is moderate, the wave is more rounded has a slow rise a peak in late systole, and an abrupt descending limb in early diastole The liver of these patients has been called the systolic liver †

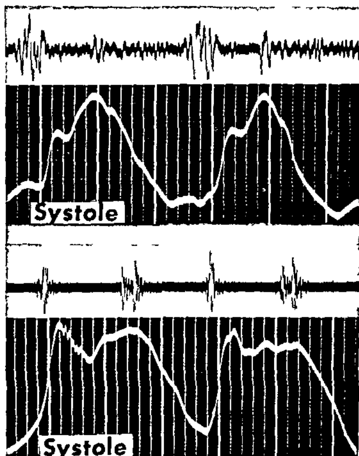


FIG 180 Two cases of rheumatic heart disease with mitral and tricuspid lesions Hepatic tracings The high systolic plateau indicates severe tricuspid insufficiency

Venous Pressure Cardiomanometry

Venous pressure is usually elevated

The pressure of the right atrium is slightly elevated and presents a typical

* It is a common misapprehension that a large right ventricle may cause a transmitted systolic pulsation of the liver which may be mistaken for that of tricuspid insufficiency Actually if there is a transmitted pulsation it takes place only in the first part of systole (early systolic impact followed by systolic depression) That caused by tricuspid insufficiency is either late systolic (slight lesion) or all systolic and plateau like (severe lesion) No mistake is possible whenever a hepatic tracing is available

† It has been stated that severe stenosis causes a delay of both the rise and the fall of the tracing This would differentiate insufficiency due to valvulitis from that due to dilatation

increase during ventricular systole This has been demonstrated both in experimental defects[7] and in clinical cases The initial tension of the right ventricle is increased but the amplitude of the ventricular pulse pressure is decreased on account of the regurgitation The pulmonary pulse pressure is decreased on account of reduced ejection of the right ventricle

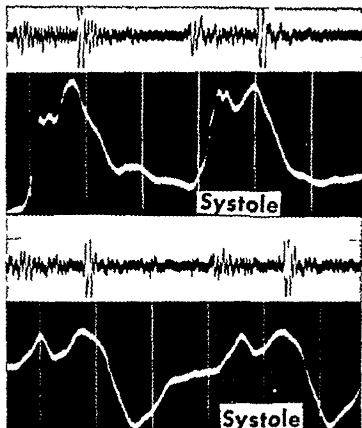


FIG 181 Rheumatic heart disease with mitral and tricuspid stenosis and insufficiency *Above* Jugular tracing *Below* Hepatic tracing High systolic plateau

Pneumocardiogram

There is a deep negative systolic wave (fusion of *v1* with *v2*) followed by a positive early diastolic wave The succession of the negative and positive waves may cause a very impressive movement of air (*cog wheel respiration*)[8, 9] (Fig 182)

Electrokymography

The border tracing of the right atrium in tricuspid insufficiency should present a typical pattern, identical to that of the left atrium in mitral insuffi

ciency Actually the right atrial tracing is so influenced by the contraction of the right ventricle that such a pattern is encountered only in severe regurgitation In many of the author s cases the liver and the jugular tracing were typical while the electrokymogram was not When typical, the eky presents a deep presystolic, negative wave caused by right atrial contraction This is followed by a large, square positive systolic plateau, caused by the blood which flows back into the right atrium during ventricular systole The plateau resembles a tracing of intraventricular pressure as it has been demon

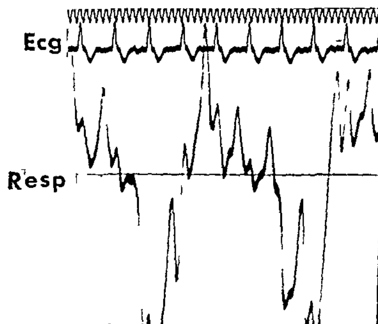


FIG 182 Cog wheel respiration in tricuspid insufficiency without stenosis (p m control) Ecg and nasal respiration

strated by Marchal and co workers [15]* The border tracing of the right ventricle shows an abrupt and early termination of contraction both in clinical cases and in experimental studies [7]

Conclusions

The graphic data having diagnostic importance are (Fig 183)

1 Evidence of a systolic murmur over the tricuspid area with inspiratory increase (phonocardiogram)

2 Evidence of increased volume of the right atrium during ventricular systole (jugular and hepatic tracings electrokymogram of the right atrium)

* As most cases present both mitral and tricuspid insufficiency special care should be taken to avoid recording a *left* atrial tracing on the right border or in a densogram

3 Evidence of high pressure within the right atrium with severe increase in systole (catheterization)

RELATIVE TRICUSPID INSUFFICIENCY

This condition is frequent especially in severe congestive failure with dilatation of the right ventricle The soft systolic murmur is recorded in phonocardiograms and is similar to that caused by an organic lesion The systolic plateau visible in jugular and hepatic tracings may not be as typical as in organic insufficiency in general one can observe a gradually rising line during ventricular systole culminating in a peak soon after the second sound *

TRICUSPID STENOSIS

Electrocardiogram

The electrocardiogram may present a high P wave However atrial fibrillation is often present

Phonocardiogram

The phonocardiogram may reveal the vibrations of a diastolic murmur over the tricuspid area Comparative tracings may show that this murmur is louder and has a lower pitch than a diastolic murmur recorded at the apex (Fig 179), moreover it increases in inspiratory apnea (Fig 178)[13]

Jugular and Hepatic Tracings

Both the jugular and the hepatic tracings show a high presystolic atrial wave if sinus rhythm is preserved (Figs 184 and 185) The large atrial wave of tricuspid stenosis described by MacKenzie[10] was confirmed in more recent years by several authors[3, 1, 18] Puddu[1] noted that if there is no venous engorgement the high presystolic pulsation is present only in the jugular bulb and not in the more distal parts of the vein This large wave has no diagnostic value because it may be present in patients having heart failure and right ventricular hypertension without tricuspid stenosis[2, 8]

Electrokymogram

The eky of the right atrium reveals an increased depth of the presystolic wave in cases with sinus rhythm (Fig 185A)

Catheterization

The pressure of the right atrium is high but there is no increase during ventricular systole

Conclusions

Graphic data of some value are revealed by the ecg and the jugular and hepatic tracings but only if sinus rhythm is preserved Even these are not absolutely pathognomonic A diastolic murmur increased by inspiration (pho

* See second footnote at page 356

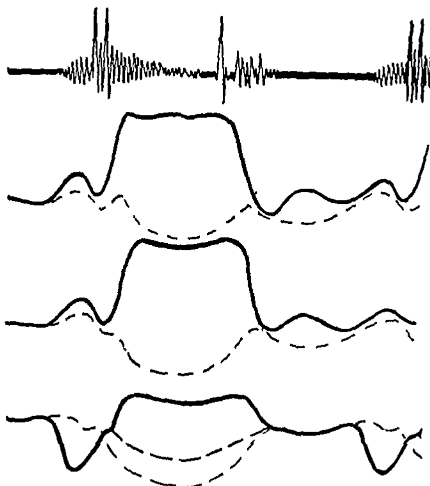


FIG 183 Scheme of the changes caused by tricuspid insufficiency Full lines = abnormal pattern Dotted lines = normal pattern Stippled line = abnormal eky in moderate insufficiency Phonocardiogram jugular and hepatic tracings, electrokymogram of the right atrium

nocardiogram) and the evidence of high right atrial pressure (catheterization) are found both in cases with sinus rhythm and in those with atrial fibrillation

RELATIVE TRICUSPID STENOSIS

This condition is caused by severe enlargement of the right ventricle with a normal tricuspid valve It may occur in cases with chronic cor pulmonale The phonocardiogram reveals the existence of a diastolic rumble which be comes louder in inspiration and, therefore simulates the rumble of organic tricuspid stenosis [14] This possibility is rare because the ostium of the tricuspid valve is usually dilated whenever there is severe right ventricular enlargement

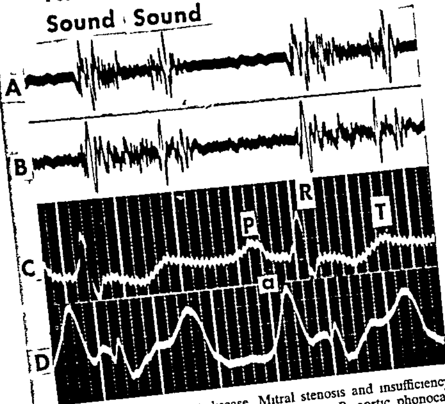


FIG 184 Rheumatic heart disease Mitral stenosis and insufficiency *tricuspid stenosis A* Pulmonic phonocardiogram *B* aortic phonocardiogram *C* electrocardiogram *D* hepatic tracing Split second sound due to delay of pulmonic valve closure High presystolic wave in the hepatic tracing (Superimposed tracings)

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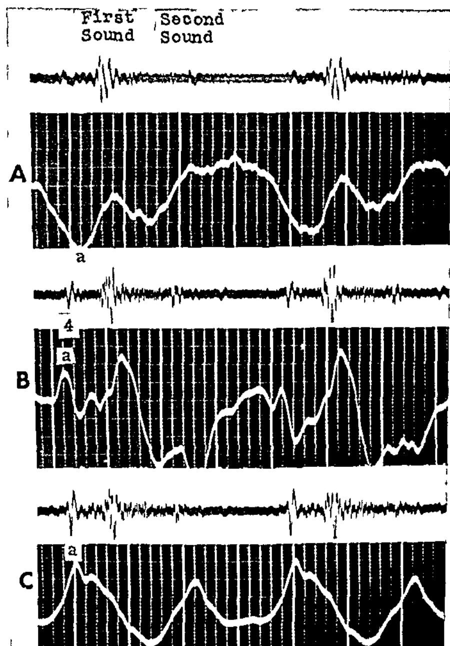


FIG 185 Rheumatic heart disease Mitral insufficiency slight tricuspid stenosis *A* Electrokymogram of right atrium deep and prolonged presystolic wave *B* Phonocardiogram triple rhythm Jugular tracing high presystolic wave *C* Hepatic tracing high presystolic wave

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CHAPTER 44

Pulmonic Valve Lesions

PULMONIC INSUFFICIENCY

Electrocardiogram

The typical finding is right axis deviation, evidence of right ventricul hypertrophy is frequent, and there may be right bundle branch block The wave is often high in leads 2 3 and aVF, and especially in V1 (pattern (right atrial hypertrophy [Fig 222])

Phonocardiogram

The phonocardiogram reveals a diastolic murmur which is similar to tha of aortic regurgitation except for the area of best recording (second an third left interspaces) (Figs 186 and 187) The vibrations of this murmu are frequently of a lower pitch than in aortic insufficiency (rougher murmur and are well transmitted both toward the xiphoid process and toward the le clavicle[4]

Pneumocardiogram

The pneumocardiogram may show a high *p wave* in systole, evidence of strong pulsation of the pulmonary arteries

Pulse Tracings

Pulse tracings fail to show the rapid rise and the rapid fall which ar typical of aortic insufficiency

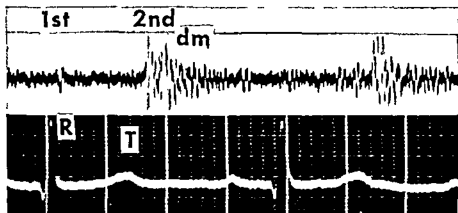


FIG 186 Phonocardiogram of pulmonic insufficiency The patient had a patent ductus with a typical murmur Following ligation without division a soft early diastolic pulmonic murmur (*dm*) appeared The pulmonic insufficiency was apparently caused by traction

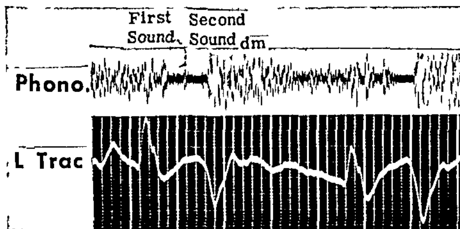


FIG 187 Mitral stenosis plus organic pulmonic insufficiency Typical early diastolic murmur (*dm*) over second left interspace Stethoscopic and linear tracings over pulmonic area

Electrokymogram

The border tracing of the pulmonary knob shows high pulsations larger than those of the aorta These pulsations have a rapid rise and a rapid collapse[2] The densograms of the hila and lungs also show large and rapid pulsations (*pulsus celer*) (Fig 188)

Cardiomanometry

The pulse pressure is increased in both the right ventricle and the pulmonary artery Diastolic pressure of the latter is extremely low and may be zero (Fig 188)

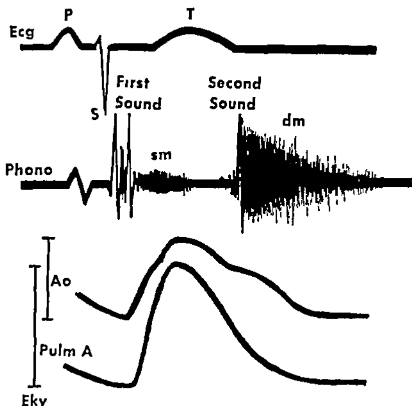


FIG 188 Scheme of the changes in pulmonic insufficiency Electrocardiogram phonocardiogram, electrokymograms of the aortic and pulmonic arches

Conclusions

The graphic data having a diagnostic value are

1 Evidence of right ventricular and atrial hypertrophy (electrocardiogram)

2 Soft early diastolic basal murmur 'in decrescendo at the left of the sternum with poor transmission (phonocardiogram)

3 Absence of a collapsing arterial pulse (pulse tracing)

4 High and collapsing pulsations of the pulmonary artery hila and lungs (electrokymography)

5 Increased pulse pressure and low diastolic pressure in the pulmonary artery (cardiomanometry)

These data are sufficient for establishing a correct diagnosis even in the presence of other valvular defects

RELATIVE PULMONIC INSUFFICIENCY

A relative regurgitation of the pulmonic valve may occur in any condition leading to severe pulmonic hypertension It has been described in acute and chronic cor pulmonale Lutembacher s syndrome and mitral stenosis In the latter the resulting murmur is known as the *G Steell murmur* In phonocardiograms, the vibrations of the murmur are usually of brief duration and of a high pitch

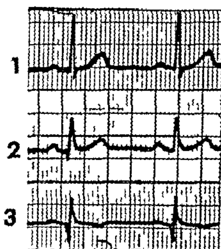


FIG 189 Case of pure pulmonic stenosis Electrocardiogram (Lack of right axis deviation)

PULMONIC STENOSIS

Electrocardiogram

The electrocardiogram shows right axis deviation There is evidence of right ventricular hypertrophy and there may be right bundle branch block The P wave is often high in leads 2 3 aVF and especially in V1 (pattern of right atrial hypertrophy [Fig 222])

Phonocardiogram

The tracings recorded over the second left interspace may show a very high vibration [3] This is the opening snap of the pulmonic valve It is followed by a series of both high and low pitched vibrations presenting first an increase and then a decrease and resembling the shape of a pulse wave This is the so called *diamond shaped murmur* (Fig 190) However the murmur is sometimes so loud that it includes all systole without any variation of intensity The second sound is weak however if the murmur is loud and prolonged, it is difficult to state whether the latter is weak or covered by the murmur

Jugular Tracing Hepatic Tracing

These tracings may show a high atrial wave

Epigastric Tracing

This tracing shows a high atrial wave and a high but slow initial ventricular wave

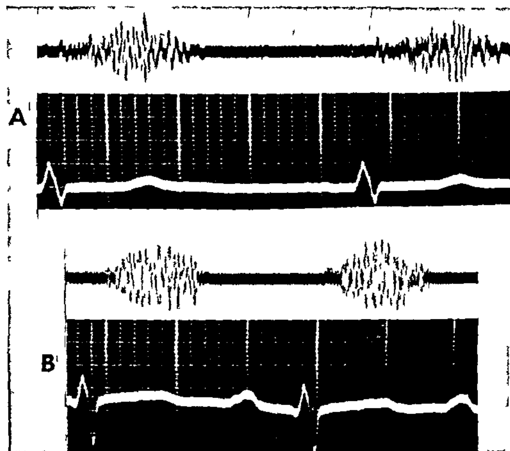


FIG 190 Pure pulmonic stenosis proven by catheterization Phonocardiograms *A* Young woman of 23 *B*, Child of 8

Apex Cardiogram

There frequently is a negative systolic wave

Electrokymogram

The border tracing of the pulmonary knob reveals frequently a decreased magnitude of the pulsations However, these may be large in cases with valvular stenosis or associated aneurysm of the pulmonary artery The pulsation is late and has a slow rise a late peak and no incisura The most typical tracings were found in cases with post stenotic dilatation [8] The densograms of the hilar shadows and especially those of the lungs reveal small or absent pulsations

Cardiomanometry

The pressure pulse of the *right ventricle* is large Pressures of 65/5 and 190/0 have been encountered [6, 7] On the other hand the pressure of the

pulmonary artery is normal or low with values of 15/0 to 30/14 or 30/18 the diastolic pressure of the latter may be slightly increased Thus the pressure gradient between right ventricle and pulmonary artery is increased (Fig 191) This is the most significant fact which has diagnostic value in cases of mild and well tolerated stenosis In some of these cases right ventricular pressure is normal and pulmonic pressure is slightly low Exercise increases the gradient Pressure tracings of the right atrium may show the existence of extremely high atrial waves due to strong contractions of the right atrium [1]

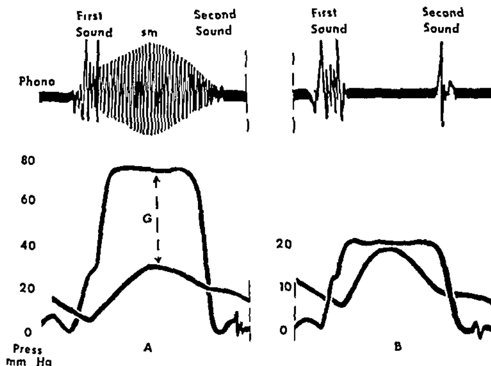


Fig 191 Scheme of the changes in pulmonic stenosis Phonocardiogram and pressure of right ventricle and pulmonary artery *A* Pulmonic stenosis *B* normal *G* gradient of pressure between right ventricle and pulmonary artery

Conclusions

The graphic data having diagnostic value are

1 Evidence of right ventricular and right atrial hypertrophy (electrocardiogram)

2 Loud diamond shaped systolic murmur and weak second sound over the second left interspace (phonocardiogram)

3 Small slow pulsations of the pulmonary artery and absence of pulsations of the lungs (electrokymogram)

4 Increased gradient of pressure between right ventricle and pulmonary artery (cardiomanometry)

The last has a decisive value in doubtful cases and in moderate stenosis

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CHAPTER 45

Uncomplicated Shunts

Several uncomplicated shunts are the result of congenital abnormalities The most common are atrial septal defects ventricular septal defects and patent ductus arteriosus Less common is a congenital fistula between aortic root and pulmonary artery Acquired shunts may be the result of myocardial infarct purulent myocarditis, or syphilis

ATRIAL SEPTAL DEFECTS

Abnormal communication between the two atria may be due to persistence of the foramen ovale other defects of the septum (including persistence of the foramen primum) or complete absence of the interatrial septum The severity of the signs increases from the first to the last of the three possibilities Dilatation of the pulmonary artery and hypoplasia of the aorta are frequently associated with these lesions

Electrocardiogram

Right axis deviation is nearly constant Right ventricular hypertrophy is frequent Right bundle branch block is very common reaching up to 95 per cent [10] The P wave was found high and peaked by Brumlik [3] normal by Wood and co workers [10] It has the type revealing right atrial enlargement (Fig 222) The P R interval is frequently at the upper limit of norm Atrial fibrillation is a common complication

Phonocardiogram

There may be no murmurs However a systolic murmur is common This is revealed by a series of both high and low pitched vibrations which are recorded best over the second or third left interspaces and frequently assume a diamond shaped aspect This murmur has been interpreted as a functional murmur caused by dilatation of the pulmonary artery and trigonoidation of the valve The second sound is loud and is frequently split The vibrations of an early diastolic murmur 'in decrescendo, caused by functional insufficiency of the pulmonic valve are recorded over the second and third interspaces in one half of the cases [10, 16] In advanced stages when heart failure sets in a functional diastolic rumble, simulating that of mitral stenosis, may be recorded over the midprecordium This was noted by the author in 3 cases where diagnosis was made only postmortem

Jugular Tracing Hepatic Tracing

If sinus rhythm is preserved, a high presystolic wave may be recorded This may lead to the erroneous diagnosis of tricuspid stenosis favored by a somewhat similar clinical picture

Roentgenkymogram Electrokymogram

Abnormal roentgenkymographic patterns in the border tracings of the atria were described in one case by Freda [6] It is possible that electrokymography may reveal typical patterns High pulsations over the pulmonary knob the hilar shadows, and the pulmonary parenchyma are also likely to be observed

Cardiomanometry

Catheterization of the heart reveals a normal pressure in the cavae and a normal or slightly increased pressure in the right atrium right ventricle and pulmonary artery This proves that there is an increase of flow with little change in pressure The typical data therefore, are supplied by determination of the oxygen saturation which is typically increased in the right atrium In some cases the catheter was passed through the atrial opening into the left atrium and even into the left ventricle

Conclusions

The graphic data having some value are

1 Right ventricular and right atrial hypertrophy Right intraventricular block (electrocardiogram)

2 Systolic murmur over the pulmonic area (phonocardiogram)

3 Increased pulsations of the pulmonary artery and hila (electrokymography)

However a definite diagnosis can be based only on the finding of a higher oxygen content of the right atrium (cardiac catheterization)

VENTRICULAR SEPTAL DEFECT

The clinical picture is caused by the absence the incomplete closure or the pathologic rupture of the ventricular septum The first two are due to congenital malformations while the third follows myocardial infarct or exceptionally rupture of an abscess When there is a small opening in the membranous part of the septum the syndrome is called *Roger s disease*

Electrocardiogram

This tracing may be normal However slight right axis deviation or right intraventricular block have been observed It is even more common to note a prolongation of the P R interval (Fig 192) or a complete a v block These abnormalities are due to associated lesions and may be absent The complete a v block has a somewhat high ventricular rate (50 75) so that it may not be noted at casual physical examination The QRS may be M shaped S T and T may be atypical Prominent Q tall R and inverted U waves in V5 V6 have been observed by Wood[16] while V1 showed well formed R waves

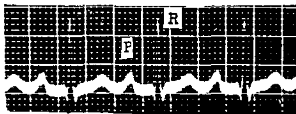


FIG 192 Electrocardiogram of a case of ventricular septal defect with long P R interval (0 22)

Phonocardiogram

The most typical finding is that of a loud systolic murmur (Fig 193) This may be recorded over the entire precordium but is usually loudest over the third left interspace It frequently spreads toward the right side of the chest and may be recorded fairly well over the right midclavicular line It may spread upwards to the aortic area It is usually much fainter at the apex even in young children This murmur may have various graphic aspects The vibrations usually are not in decrescendo like those of a mitral murmur neither are they diamond shaped like an aortic murmur They start in early systole and may present rhythmic variations in loudness like organ pipes [13] There may be a *late systolic murmur* with an abrupt end at the second sound The second pulmonic sound is usually loud but is seldom split There frequently is a triple rhythm occasionally quadruple rhythm can be observed In certain

cases a soft early diastolic murmur (aortic?) has been noted Confusion with the murmur of patent ductus is then avoided only by a careful study of the sound tracing

Low Frequency Tracing

According to Viciu [1] the low frequency tracing reveals an extremely short duration of the isometric tension period due to the shunt The tracing becomes normal again during a Valsalva test

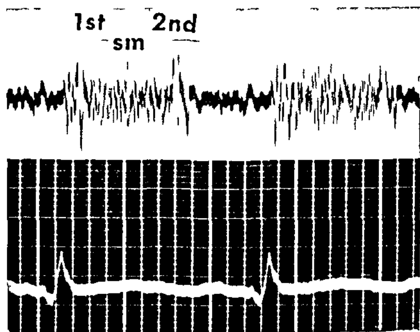


FIG 193 Phonocardiogram at midprecordium in a case of ventricular septal defect confirmed by catheterization Loud all systolic murmur

Electrokymography

Observations of the author with Fleischner[9] revealed the following data

1 The pulsations of the pulmonary artery are higher than normal but still smaller than those of the aortic arch The rise of the pulmonic pulse is normal

2 The pulsations of the hila and lungs are somewhat higher than normal the rise of the pulse is slightly faster than normal (tendency to pulsus celer)

3 The speed of the pulse wave between pulmonic arch and pulmonary parenchyma is normal

Cardiomanometry

The pressure is normal in the right atrium, it may be normal in the right ventricle and pulmonary artery On the other hand it is frequently increased

in both chambers on account of increased pulmonary peripheral resistance[14] Oxymetry reveals a typical increase of oxygen saturation in the blood of the right ventricle

In the acquired cases the murmur is very loud the electrocardiogram indicates right ventricular strain cardiomanometry reveals increased right ventricular pressure

Conclusions

In most cases a graphic diagnosis can be based on the following data

1 Normal or slightly abnormal electrocardiogram (in acute rupture right axis deviation) Possible prolongation of P R or complete a v block

2 Typical phonocardiogram with evidence of a rough and long systolic murmur well transmitted toward the right side of the chest

3 Electrokymographic evidence of normal speed of the pulse wave in the pulmonary circulation

In doubtful cases oxymetry reveals increased oxygen content in the right ventricle (cardiac catheterization)

PATENT DUCTUS ARTERIOSUS

Electrocardiogram

In certain cases this tracing is normal (Fig 194) However it is common to observe left axis deviation (Fig 195) Left bundle branch block may be present Evidence of left ventricular hypertrophy may be noted in severe cases A slight prolongation of the P R interval has been described in some of the cases it was probably due to an associated lesion

Phonocardiogram

The phonocardiogram gives important data There is a typical *machinery murmur* revealed by coarse vibrations These start at the beginning or at the middle of systole increase during the last part of systole cover the second sound and then decrease gradually during the first part of diastole In other words there is a single late systolic–early diastolic murmur overriding the second sound (Fig 196) This murmur different from any other caused by valvular lesions was described by Routier[12] and confirmed by Hubbard[7] In cases with a large ductus the murmur is continuous but still increases in late systole In young children clinically one may have the impression of a systolic murmur The tracing however reveals that the murmur has still the maximum in late systole and early diastole

The murmur has the above-described aspect because it is caused by the blood passing through the ductus If the ductus is narrow the blood passes only during the phase of maximum height of pressure in the aorta and lowest pressure in the pulmonary artery If the ductus is wide blood passes through

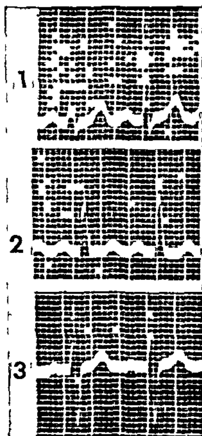


Fig 194 Left axis deviation in a case of patent ductus arteriosus

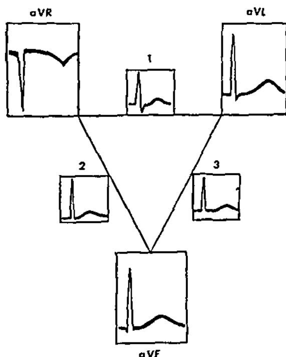


Fig 195 Scheme of the electrocardiogram from a case of patent ductus without axis deviation

out the entire cardiac cycle but still has an acceleration in late systole and continues in early diastole, when the pressure of the pulmonary artery drops to a very low level This murmur is recorded best over the first and second left interspaces and fairly well over the suprasternal notch and the second right interspace The author has frequently noted a loud snapping sound in early diastole without being able to explain its exact nature it may be a split second sound or a special vibration of the wall of the pulmonary artery A functional diastolic rumble may be recorded at the apex [11]

Cardiomanometry

Normal pressure is found in the right atrium The pressure of the right ventricle is either normal or increased That of the main pulmonary artery is increased in two thirds of the cases [16] Whenever pulmonary arterial resistance is increased the level of pulmonary pressure is proportional to the size of the shunt otherwise there may be a large shunt with a normal pulmonary pres

sure An abrupt rise of both systolic and diastolic pressures and a characteristic pulse contour at the site of the ductus have been described [8] Oxymetry reveals a typically increased oxygen saturation in the blood of the main pulmonary artery

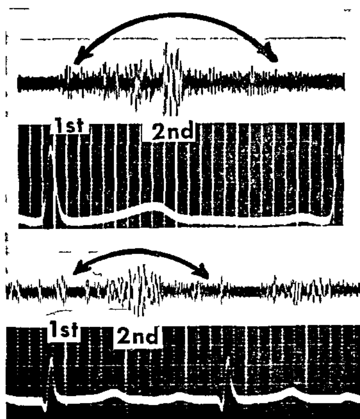


FIG 196 Phonocardiograms of two cases of patent ductus arteriosus Pulmonic area Continuous murmur overriding the second sound Maximum in late systole and early diastole

Electrokymography

The most significant results are obtained by the study of the border tracings of the aortic and pulmonic knobs and of the densograms of the hila and lungs (Fig 197) [9]

1 The pulsations are increased in both the aorta and the pulmonary artery more so in the latter There is a rapid rise of the pulse of the pulmonary artery (*pulsus celer*) and there may be a high dicrotic wave *

* Older studies of roentgenkymography described a double wave the second wave was attributed to the blood shunted through the ductus It is difficult to ascertain whether it is so or whether the dicrotic wave is increased

2 The pulsations of the hila and lungs are many times larger than in normal subjects the rise of the pulse is rapid (*pulsus celer*), a high dicrotic wave or a systolic plateau are recorded over both structures

Comparison of the three tracings reveals an increased speed of transmission of the pulse wave

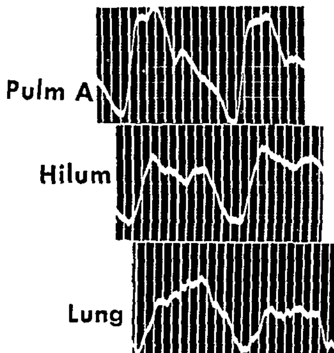


Fig 197 Electrokymograms in a case of patent ductus Rapid pulse and high dicrotic wave in the pulmonary artery The high pressure flow through the ductus creates a plateau like pattern in the hilum and lung Increased speed of transmission of the pulse wave (Tracings superimposed so that they preserve their exact time relationship)

Pulse and Blood Pressure Tracings

In the typical cases with a large shunt the arterial pulse has a rapid rise and a rapid collapse (*pulsus celer*) Systolic pressure is slightly increased diastolic pressure is decreased the pulse pressure is increased

Conclusions

In most of the cases diagnosis can be based on the following data

1 Normal electrocardiogram or evidence of left axis deviation

2 Typical machinery murmur late systolic–early diastolic murmur overriding the second sound (phonocardiography)

3 Large and rapidly rising pulsation high dicrotic waves and increased speed of the pulmonary waves (electrokymography)

A high oxygen content and possibly a higher pressure are found in the pulmonary artery (catheterization) However this procedure is necessary only in difficult or atypical cases

AORTIC SEPTAL DEFECT

This congenital malformation presents similar data to those of patent ductus arteriosus However the murmur is recorded best over the center of the heart Differentiation of this lesion from patent ductus arteriosus is important However the only graphic differential data are a different location of the murmur finding of a high oxygen content in a more proximal section of the pulmonary artery possibility of passing the catheter into the ascending aorta

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CHAPTER 46

Complex Congenital Malformations of the Heart

These malformations are usually accompanied by the three clinical signs of cyanosis clubbing of the fingers and polycythemia They have a short circulation time due to passing of the chemical through the shunt from right to left In considering these forms as a whole one should keep in mind that the syndrome of Fallot (or tetralogy of Fallot) is the most common of the cyanotic syndromes (probably 75 per cent of the cases) and that it becomes even more statistically predominant after adolescence

These malformations are caused by an arrested development at an early stage of fetal life before completion of the torsion of the primitive cardiac tube (so called detorsion of Spitzer) The resultant abnormality consists of the stenosis of one ostium or valve plus one or more shunts dextroposition of the aorta or transposition of the large vessels The most common stenosis is that of the pulmonary ostium and it is most frequently associated with a large aorta overriding an opening of the ventricular septum It may be associated with an additional atrial septal defect Stenosis (or atresia) of the tricuspid valve is next which is compatible with life only if there is a double shunt The insufficiency of the tricuspid valve caused by a special malformation is usually associated with an atrial septal defect and another shunt

Stenosis of the aorta (or coarctation of the aorta*) is usually associated with patency of the ductus

Various graphic findings are common to several of these malformations

Electrocardiogram[5 6 8 10]

Right ventricular hypertrophy and ischemia are common (Fig 198) They are found in

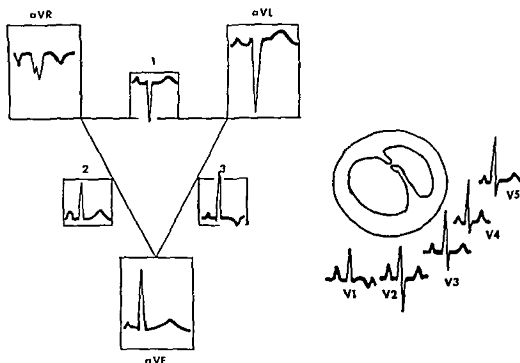


FIG 198 Scheme of the electrocardiogram from a case of tetralogy of Fallot

1 Tetralogy of Fallot (including cases with pulmonic atresia)
2 Transposition of the large vessels
3 Pulmonic stenosis plus atrial septal defect (frequent but not constant)
4 Patency of the ductus plus aortic stenosis or atresia or coarctation of the aorta (infantile type)
5 Mitral atresia (or severe stenosis) plus atrial septal defect and patent ductus
6 Eisenmenger complex
7 Lutembacher s syndrome

There may be *right bundle branch block* The P wave has a high voltage in leads 2 3 and aVF it is high and diphasic with initial and predominant positive phase in V1 (Fig 222) indicating right atrial hypertrophy

Left ventricular hypertrophy and ischemia are less common They are found in

* Uncomplicated coarctation is described in Chapter 51

1 Tricuspid atresia (or severe stenosis) plus atrial septal defect and patent ductus arteriosus

2 Ebstein s syndrome (tricuspid insufficiency plus patent foramen ovale) *

3 Common arterial trunk (except rarer cases)

4 Single ventricle with a common arterial trunk single ventricle with both great vessels arising from a rudimentary chamber (cor biloculare or cor triloculare biatriatum)

5 Pulmonic atresia (or severe stenosis) plus atrial septal defect and patent ductus arteriosus

6 Pentalogy of Fallot (tetralogy plus foramen ovale)

In many of these cases, there also is *left bundle branch block* There frequently is evidence of *right atrial hypertrophy* (Fig 222) contrasting with *left ventricular hypertrophy*

In deciding about changes of the axis it will be necessary to exclude positional changes revealed by the unipolar limb leads and to observe the data supplied by the unipolar chest leads including V3R and V4R (p 233)

Prolonged P R interval (or complete a v block) may occur in any of the complex malformations but is more common in those including a ventricular septal defect

Phonocardiogram

The phonocardiogram gives typical data only in a few complex malformations

1 In malformations including patency of the ductus there is a late systolic–early diastolic murmur overriding the second sound (p 375)

2 In malformations including an overriding aorta (like the tetralogy of Fallot or the syndrome of Eisenmenger) different pictures are recorded over the various areas A systolic murmur with various possible configurations most often having a diamond shaped aspect is recorded over the left border of the heart (pulmonic stenosis) (Figs 190 and 191) On the other hand a triple rhythm due to addition of an early systolic group of vibrations is frequently recorded over the center of the heart or at the right of the sternum (Fig 199) This typical picture first described by Calo[3] was found by the author in several cases It may be explained with a vibration of the rim of the septal foramen caused by the meeting of the two ventricular streams

3 Cases of Lutembacher complex may present a diastolic rumble and an opening snap over the midprecordium similar to those of uncomplicated mitral stenosis (p 326)

4 Cases with an Eisenmenger complex may present not only a systolic murmur but also a high pitched early diastolic murmur over the pulmonic

* In Ebstein s syndrome bundle branch block or intraventricular block is extremely common This may mask the evidence of ischemia

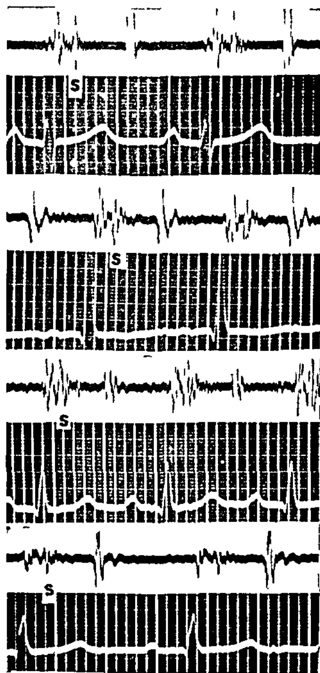


Fig 199 Phonocardiogram in four cases of tetralogy of Fallot Triple rhythm due to a systolic group of vibrations (so-called systolic gallop), s s=systolic vibrations

area This is caused by pulmonic insufficiency (relative or absolute) which is a common complication (Figs 186 and 187) The second pulmonic sound is loud and may be split

5 Cases having a foramen ovale in association with pulmonic stenosis tricuspid atresia or tricuspid insufficiency (the last combination is called Ebstein s syndrome) may present a short presystolic murmur separated from the first sound This murmur was recorded by the author at the left of the sternum (second and third interspaces) and seems due to passing of the blood through the shunt during atrial contraction

All other cases reveal the vibrations of a systolic murmur and this may vary in configuration and loudness over the various areas according to the combination of lesions

It should be noted that the phonocardiogram may exclude rheumatic heart lesions or on the other hand reveal such a typical picture of mitral insufficiency and stenosis that the diagnosis may be changed to that of rheumatic heart disease In this regard two considerations should be kept in mind

1 That cases with an atrial septal defect plus a congenital stenosis of the tricuspid or a rheumatic stenosis of the mitral do present a diastolic rumble caused by a v valvular stenosis

2 That a relative stenosis of either the mitral (p 333) or the tricuspid valve (p 360) may cause low pitched diastolic vibrations which should be differentiated from those of an organic stenosis

Jugular Tracing Hepatic Tracing

These tracings present a certain interest in the combination of tricuspid insufficiency with an atrial septal defect (Ebstein s syndrome) This syndrome is caused by a special malformation of the tricuspid valve which causes lack of apposition of the leaflets and results in the fact that one part of the right ventricle being above the valve pushes the blood backwards during ventricular systole For these reasons both tracings present a typical *systolic plateau identical to that of tricuspid insufficiency (Figs 181 183) (p 355)* In the differential diagnosis one should keep in mind that in exceptional cases of *Lutembacher s syndrome a high positive pulse was recorded over the jugular* veins it was caused by the blood regurgitating through the mitral valve and *passing from the left to the right atrium through the septal defect* This event is however rare The jugular tracing may be absolutely normal in cases of *tetralogy of Fallot (Fig 200)*

Carotid Tracing

The carotid tracing presents interest in the following conditions

1 Those associated with *aortic stenosis* an anacrotic staggered pulse is recorded (Figs 174 and 176) (p 346)

2 Those associated with a *large overriding aorta* (Fallot Eisenmenger) a high carotid pulse with normal profile is recorded (Fig 200)

Electrokymogram

The electrokymogram reveals many important data in complex malformations Its value is not as yet completely proven simply because few cases have been studied so far

Typical data are the following

1 *Cases with pulmonic stenosis* (Fallot and others) Small late staggered pulmonic pulse in certain cases lack of any arterial pulsation in the pulmonic window recording of a normal atrial pattern due to the left atrial appendage in others [13] small pulsations of the hilar shadows no pulsation of the pulmonary parenchyma (p 199) This is particularly important in those cases where fluoroscopy reveals large hilar vessels receiving blood from the bronchial or mediastinal arteries these vessels have little or no pulsation The tracing of the right ventricle reveals a powerful systolic contraction

2 *Cases with pulmonic dilatation* (Eisenmenger) present large pulsations of the pulmonary artery hila and lungs If the pulmonary artery is severely dilated (aneurysm), the tracing reveals a slow rise a rounded peak, and a slow return to the baseline

3 *Cases with pulmonic insufficiency* present a typical pattern over the pulmonary arch (p 375)

4 *Cases with coarctation of the aorta* present sudden cessation or decrease of the pulsations below the narrow section

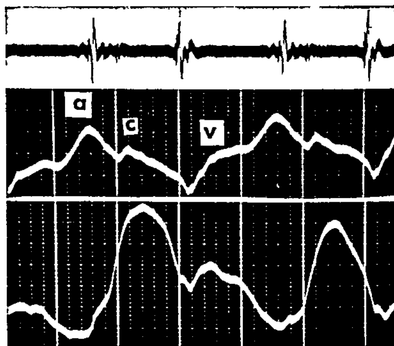


Fig 200 Phonocardiogram jugular and carotid tracings in a case of tetralogy of Fallot (superimposed tracings)

5 *Cases with mitral regurgitation* (*Lutembacher s syndrome*) present a typical pattern over the border of the left atrium which resembles a tracing of intraventricular pressure (p 321) (Figs 149 150 and 151) It is possible that in some cases a similar pattern may be found over the right atrial border (regurgitant wave through atrial septum) However, this was not true in 3 cases studied by the author The tracings of the pulmonary artery, hila and lungs reveal powerful pulsations That of the pulmonary artery may reveal a slow rise and a rounded profile in cases with pulmonic aneurysm

6 *Cases of Ebstein s syndrome* may reveal a plateau like tracing of the right atrium indicating severe tricuspid regurgitation (p 357) (Fig 183)

Cardiomanometry

It is difficult to overemphasize the value of cardiac catheterization in complex malformations of the heart It should be kept in mind that the data supplied by cardiomanometry are supplemented by those of oxymetry and by the fact that the catheter may penetrate certain structures in an unusual way or may not be able to enter passages which are normally open Thus passing of the catheter from the right ventricle into the aorta reveals a riding aorta while failing to penetrate the pulmonary artery may indicate pulmonic stenosis Penetration from the right into the left atrium indicates atrial septal defect from the right into the left ventricle ventricular septal defect Penetration from the pulmonary artery into the aorta confirms patency of the ductus Lack of penetration into the right ventricle may be caused by tricuspid atresia

Pressure changes revealed by catheterization are the following

1 *High pressure in the right atrium is found in*

a Pulmonic stenosis plus patent foramen ovale
b Tricuspid stenosis or atresia with shunts
c Tricuspid insufficiency plus patent foramen ovale (Ebstein s syndrome)
d Mitral stenosis and insufficiency plus patent foramen ovale (Lutembacher s syndrome)

In (c) and (d) but especially in (c) there is a marked increase of pressure during ventricular systole due to regurgitation

2 *High pressure in the right ventricle* is found in

a Tetralogy of Fallot
b Eisenmenger s complex
c *Transposition of the large vessels*
d Pulmonic stenosis plus patent foramen ovale
e Cor biloculare and cor triloculare with a single ventricle
f Common arterial trunk
g Tricuspid insufficiency plus atrial septal defect (Ebstein s syndrome)
h Aortic stenosis or coarctation plus patency of the ductus
i Mitral atresia with double septal defect

3 *High pressure in the pulmonary artery* is found in

a Eisenmenger s complex
b *Aortic stenosis or coarctation plus* patency of the ductus
c Transposition of the large vessels (the catheter may enter the pulmonary *artery only after penetrating into the left ventricle through the patent ventricular septum*)
d Lutembacher s syndrome

e Cor biloculare and cor triloculare with a single ventricle
f Mitral atresia with double septal defect

Correlation

The following schematic data can be useful in some of the most common conditions

1 *Tetralogy of Fallot* Evidence of right ventricular dilatation and hyper trophy (ecg) Systolic pulmonic murmur, weak second pulmonic sound, systolic gallop (phono) Small and abnormal pulsations of the pulmonary artery or lack of any pulse, no pulsation in the lungs (eky) High pressure in the right ventricle, possible penetration of the catheter into the left ventricle or aorta difficult penetration into the pulmonary artery, increased pressure gradient between right ventricle and pulmonary artery due to high pressure in the former and normal or low pressure in the latter

2 *Eisenmenger s complex* Evidence of right ventricular hypertrophy and strain (ecg) Systolic gallop, systolic and possible early diastolic pulmonic murmur loud and frequently split second pulmonic sound (phono) High pressure in the right ventricle and pulmonary artery easy penetration of the catheter into the latter, possible penetration into the left ventricle or aorta

3 *Lutembacher s syndrome* Evidence of a diastolic rumble and presystolic murmur over midprecordium (phono) Large pulsation of the pulmonary artery hila, and lungs typical pattern of regurgitation over the left atrium (eky) High pressure in the right ventricle and pulmonary artery (catheterization)

4 *Ebstein s syndrome* Evidence of left ventricular hypertrophy and strain and of right atrial dilatation (ecg) Systolic tricuspid murmur (phono) Evidence of tricuspid regurgitation (jugular and hepatic tracings) High pressure in the right atrium and ventricle possible penetration of the catheter into left atrium

5 *Tricuspid stenosis or atresia with shunts* Evidence of left ventricular and right atrial hypertrophy and strain (ecg) High pressure in the left atrium difficult or impossible penetration of the catheter into the right ventricle

6 *Aortic stenosis or coarctation plus ductus* Evidence of right ventricular hypertrophy and strain (ecg) Typical murmur riding over the second sound (phono) High pressure in the chambers of the right heart and pulmonary artery possible penetration of the catheter through the ductus into the aorta

7 *Transposition of the large arteries* Evidence of right ventricular hypertrophy (ecg) Possible penetration of the catheter into the aorta impossible penetration into the pulmonary artery from the right ventricle

8 *Common trunk* Evidence of left ventricular and possibly right atrial

hypertrophy and strain (ecg) Possible penetration of the catheter into the trunk or aorta

9 *Pulmonic stenosis with patent foramen ovale* Evidence of right ventricular hypertrophy (occasional left) (ecg) Diamond shaped systolic murmur over pulmonic area weak second pulmonic sound (phono) High pressure in the chambers of the right heart difficult penetration of the catheter into the pulmonary artery, if successful penetration evidence of low pulmonic pressure with increased gradient between right ventricular and pulmonic pressures possible penetration into the left atrium

Conclusions

Diagnosis of complex malformations is difficult on account of multiple possibilities and individual variations All possible data are necessary for a correct diagnosis and they include physical and fluoroscopic data, electrocardiographic phonocardiographic and electrokymographic tracings Both angiocardiography and catheterization supply fundamental data those of the latter are based on cardiomanometry oxymetry and penetration of the catheter All of them may be necessary in difficult or unusual cases

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CHAPTER 47

Disturbances of the Cardiac Rate and Rhythm

DISTURBANCES DUE TO ABNORMAL EXCITABILITY

Sinus Tachycardia

Sinus tachycardia may be caused by exercise digestive disturbances or emotion It may be due to fever drug action or endocrine disorders Many cardiac diseases may be associated with it low blood pressure is frequently a cause of tachycardia

ELECTROCARDIOGRAM The atrial and ventricular waves follow each other in a normal way P R may be slightly longer but not over 0 20 second Diastole is short so that the end of T is near the following P (Fig 201 A) In the adult the rate varies between 80 and 150 but is seldom above 100 Occasionally higher figures have been described

JUGULAR TRACING There may be fusion of v with the following *a* wave

PHONOCARDIOGRAM The heart sounds are short and loud A triple rhythm is not unusual because of tumultuous ventricular filling

Sinus Bradycardia

This is due to increased excitability of the vagus nerve reflex vagal stimulation or depression of the s a node Endocrine disorders may cause sinus bradycardia

ELECTROCARDIOGRAM The electrocardiogram is normal and shows a long

diastole P R is normal The rate is usually between 65 and 50, and only exceptionally below 50 (Fig 201 B)

PHARMACOLOGIC AND FUNCTIONAL TESTS The most important is the *atropine test* Injection of 1 mg of atropine sulfate hinders the action of the vagus nerve and accelerates the heart rate Another pharmacologic test is represented by the *inhalation of amyl nitrite* This is particularly effective in patients with coronary heart disease and bradycardia caused by functional depression of the s a node

Changes of position exertion and deep respiration usually are sufficient to accelerate the pulse in persons with sinus bradycardia Compression of the

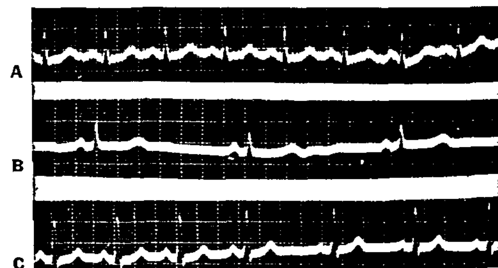


FIG 201 Electrocardiograms of *A* sinus tachycardia *B*, sinus bradycardia *C*, sinus arrhythmia

carotid sinus may arrest the heart in patients with hypersensitive carotid receptors Both this maneuver and the compression of the eyeballs may arrest the heart in patients with an excitable vagus nerve

Sinus Arrhythmia

This common irregularity of the pulse is usually associated with respiration It is particularly evident in children, convalescents or people with unusually high excitability of the vagus nerve It is also frequently present when pleuropericardial adhesions are stretched by respiratory movements It may occur in old people with arteriosclerotic lesions and coronary heart disease

Whatever the cause this type of irregular pulse is always favored by high excitability of the vagus nerve *

ELECTROCARDIOGRAM The electrocardiogram has normal waves it shows in general gradual changes in the length of diastole (Fig 201 C) If there are sudden changes and the long diastole is equal to twice the P P interval the cause is s a block

ARTERIAL TRACING The pulse waves become gradually larger and have a lower starting point then become smaller again with a higher starting point This indicates higher systolic and lower diastolic pressures during the phase of slower pulse, usually during the second half of expiration

ABNORMAL RHYTHMS 1 DISTURBANCES OF RATE AND RHYTHM CAUSED BY INCREASED EXCITABILITY OF THE MYOCARDIUM

Atrial and Nodal Tachycardia

This paroxysmal syndrome is common and may occur at any age It may be present at birth Endocrine disorders (hyperthyroidism and in general dis orders associated with uterine myoma puberty menopause or pregnancy) favor these types of tachycardia Normal individuals with the Wolff Parkinson White syndrome are prone to such attacks of tachycardia Nervous reflexes due to cardiac or extracardiac stimulation and reaching the heart along the sympathetic pathways may be involved

Paroxysmal tachycardia is due to the rapid and regular production of stimuli in some area of the atrial or septal myocardium Two different theories have been advanced (1) that of circus movement (2) that of an abnormal pacemaker in the atrial wall (parasystole) Present evidence confirms the second theory because multiple studies prove that there is no circus movement in the atrial walls

ELECTROCARDIOGRAM During the attack the ventricular complexes are reg ular and have a rate of 160–180 (occasionally higher or lower) The atrial waves have the same rate and are either upright with abnormal contour or in verted The abnormal P may precede follow or be masked by QRS it may fuse with the preceding T (Fig 203 A) When P precedes QRS there is *atrial tachycardia* when P is inverted and near QRS there is *nodal tachycardia*

* According to Froment and Gallavardin sinus arrhythmia may have different mechanisms

Sinus Arrhythmia
- Vagal
  - respiratory
  - anoxic (bulbar anoxia)
  - independent
- Myocardial
  - incomplete s a block
    - type I (periodic)
    - type II (intermittent)
  - s a standstill

The myocardial forms of sinus arrhythmia are described on pp 410–411

Cases having a faster atrial rate and a v block have been described As the mechanism of production of atrial tachycardia is similar to that of atrial flutter, it is preferable to diagnose such cases as atrial flutter (p 395)

After the attack, the ecg becomes perfectly normal However, in some cases, inversion of T lasting for three to twenty days, may be observed (post tachycardial syndrome)

JUGULAR TRACING There is only one high wave, due to fusion of the three normal waves If the atrial and ventricular contractions occur at the same time this venous wave becomes high



FIG 202 Electrocardiogram and phonocardiogram during atrial tachycardia

ARTERIAL PULSE The pulse becomes small during the attack The dicrotic wave may be high and its fusion with the following pulse wave may simulate an anacrotic pulse The speed of the pulse wave is often reduced

PHONOCARDIOGRAM The heart sounds are of short duration The interval between the first and the second sound during the attack is shorter than between the attacks (shorter systole), in one case observed by the author the length of ventricular systole was 0 16 second during the attack as compared to 0 24 second after it

The heart sounds are louder during the attack especially the first (Fig 202) In nodal tachycardia additional atrial vibrations [6] may simulate splitting of the first sound The vibrations of a functional systolic murmur may be present during the attack

CONCLUSIONS The diagnosis of atrial tachycardia is often possible on the

basis of the history The electrocardiogram taken between the attacks reveals a normal tracing while during the attack it shows a simultaneous increase of the atrial and ventricular rates In doubtful cases compression of either carotid sinus or of the eyeballs should be attempted Even if these maneuvers

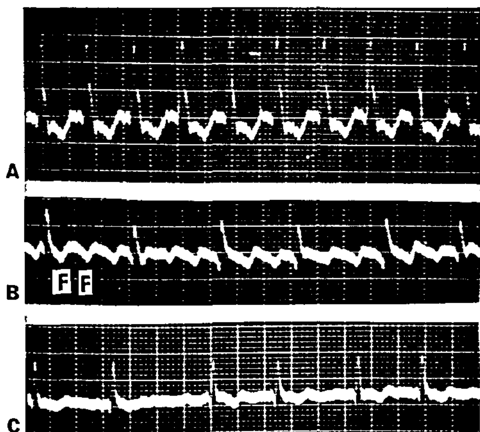


FIG 203 Electrocardiograms of *A* atrial tachycardia *B* atrial flutter (*F F*= atrial waves) *C* atrial fibrillation

do not terminate the attack they slow down the rate to a considerable extent and give evidence that there are no atrial waves between the ventricular complexes as in atrial flutter

Atrial Flutter

Atrial flutter is a persistent and abnormal condition of the atria revealed to the patient by acute attacks of rapid ventricular action Atrial flutter is found more commonly with heart disease than in its absence It may be associated with valvular defects (especially mitral stenosis) hypertension coronary heart disease or hyperthyroidism It may be found at any age

The mechanism of the disturbance is based upon

1 An abnormal production of atrial stimuli, causing regular and extremely rapid atrial contractions
2 A protective block between atria and ventricles
3 Sudden disappearance of the block, causing paroxysms of rapid ventricular contractions

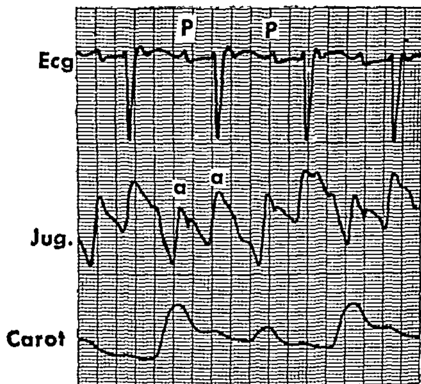


FIG 204 Atrial waves in the jugular tracing of atrial flutter (2 1) Electrocardiogram in V1 jugular tracing (amplified) brachial tracing

The abnormal production of stimuli was attributed to a circus movement involving the sinoatrial node However, experimental and clinical evidence has accumulated through the years until the experiments of Scherf,[53] further confirmed by those of Prinzmetal[4] have conclusively proven the existence of a single focus in the atrial wall

ELECTROCARDIOGRAM Between attacks of rapid pulse when the patient however, still has atrial flutter the ecg shows regular ventricular complexes of normal appearance Three or more P waves are present between them at regular intervals * The rate of the atrial waves is from 200 to 400 per minute During an attack of rapid pulse the ecg shows 3, 2 or even only 1 P wave

* When the P waves have a negative and a positive phase the negative phase corresponds to an inverted P the positive phase to a Ta wave [40]

between the QRS complexes The T waves may be deformed by fusion with the P waves Tracings recorded in V1 and V2 usually give the best evidence of the atrial waves However in certain cases leads 3 and aVF supply this evidence better (Fig 203 B) [11]

FUNCTIONAL TESTS Compression of the eyeballs or of either carotid sinus may slow down the ventricular rate which becomes a fraction of the previous rate

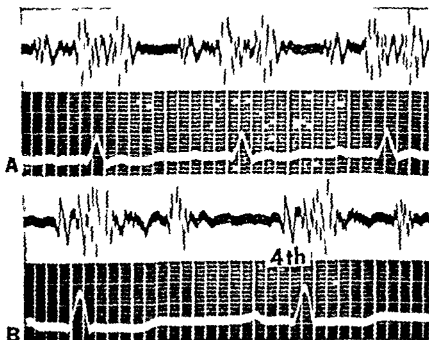


FIG 205 Phonocardiograms of atrial flutter *A* diastolic rumble during the attack *B* atrial (fourth) sound after reestablishment of sinus rhythm

CARDIOGRAM Atrial waves may be recorded in the intervals between the ventricular waves of the cardiogram [11]

JUGULAR TRACING Regular atrial waves were recorded by several workers [3, 1, 6, 38, 4, 5, 9] More recently Contro[11] has recorded high atrial waves in 5 cases of flutter by means of a linear microphone and an amplifier These give evidence of the coordinated contractions of the atria (Fig 204)

PHONOCARDIOGRAM The phonocardiogram may reveal the existence of sounds of atrial origin, not only during ventricular diastole but also during ventricular systole [6, 11, 19, 0, 1, 4, 30, 3] When the ventricular rate is rapid a functional diastolic rumble may appear at the apex simulating that of mitral stenosis (Fig 205 A) [2, 37] If slight irregularities of the ventricular rate occur a changing intensity of the first heart sound may be observed [19, 4]

ELECTROKYMOGRAM If the slit of the pickup is placed across the border of either atrium, good evidence of atrial contractions can be secured [11, 5, 33, 31] The atrial waves are usually of large amplitude and are important in cases where the electrocardiographic tracing had led to admission of flutter fibrillation (Fig 206)

CONCLUSIONS In general diagnosis of atrial flutter is made by electrocardiography Special attention should be paid to leads aVF and V1 V2 for recognition of atrial waves In doubtful cases demonstration of atrial sounds by the phonocardiogram and of atrial waves by the jugular tracing or the electrokymogram helps in excluding atrial fibrillation

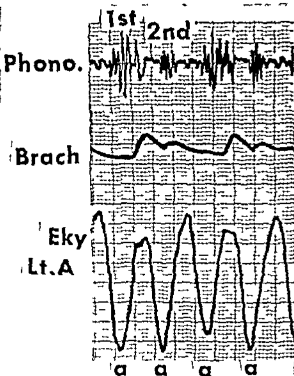


FIG 206 Electrokymogram of left atrial appendage in atrial flutter Heart sounds brachial pulse eky Rate 211

Atrial Fibrillation

This disturbance was described simultaneously by Lewis [7] and by Rothberger and Winterberg [48] It is a common disturbance of atrial function and is revealed clinically by total and complete irregularity of the ventricular contractions and of the pulse

Atrial fibrillation may occur in any case of heart disease However two types are the most common

1 Atrial fibrillation in patients with chronic distention of the atria resulting from such conditions as *mitral stenosis* combined lesions of the mitral and tricuspid valves or atrial septal defect The fibrillation is frequently associated with heart failure

2 *Atrial fibrillation* in patients with coronary heart disease and fibrosis of the myocardium The fibrillation is often present without congestive failure

Atrial fibrillation is frequently encountered in thyrotoxicosis acute myocarditis, or after coronary occlusion

Experimental conditions favoring atrial fibrillation are (1) distention or anoxemia of the atria (2) acute lesions of the atrial myocardium (3) direct or reflex stimulation of the vagus nerve and (4) digitalis poisoning

Atrial fibrillation has been the object of heated discussions between the school of Lewis and that of Rothberger The former attributed the fibrillation to a circus movement the latter to multiple foci of excitation within the atrial walls The experimental studies of Scherf [51, 53] further confirmed by Prinzmetal [45] prove that the second theory is true

ELECTROCARDIOGRAM The following data are typical there is no P wave the intervals between the various ventricular complexes are never alike having different lengths even when at first glance the ventricular rhythm seems regular careful measurements reveal the arrhythmia

There are two varieties

1 There are fine and irregular waves at a rate of 300–500 per minute This is typical atrial fibrillation (Fig 203 C)

2 There are coarse and somewhat irregular waves This has been called *coarse fibrillation* or *flutter fibrillation*

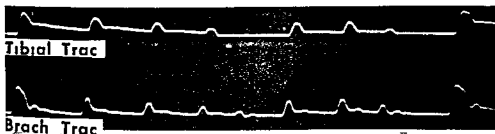


FIG 207 The arterial pulse of atrial fibrillation Complete irregularity of the intervals between waves and of the height of the pulse waves

JUGULAR TRACING The *c* and *v* waves follow each other with complete irregularity There are no *a* waves

LOW FREQUENCY TRACING The waves caused by ventricular contraction are of various height and configuration When diastole is short the subsequent period of systolic tension is longer The height of the waves varies somewhat but is not proportional to the length of the preceding diastole

ARTERIAL TRACINGS HEMODYNAMICS The pulse waves are typically irregular having various height length and configuration Patients in congestive failure often have a pulse deficit because some of the ventricular contractions are not followed by a pulse wave Moreover some waves may be present in the arteries nearer the heart and absent in those more distant [32] When failure is severe a high pulse wave may be followed by gradually smaller ones (*Galenus pulse*) (Fig 207) As long as the conditions of the myocardium are good the stroke volume is directly proportional to the length of the preceding diastole and inversely proportional to the stroke volume of the preceding cycle If however there is cardiac failure no rule can be established The

speed of the small pulse waves is always greater than that of the large waves [1]

PHONOCARDIOGRAM The heart sounds have vibrations of similar amplitudes The interval between QRS and the first sound complex may vary being longer after a short diastole as has been shown by the author (Fig 208) [31] This is more often the case in patients with mitral stenosis, the longer interval seems due to delayed closing of the mitral valve and delayed beginning of the sound on account of smaller atrioventricular gradient of pressure [2, 83]

ELECTROKYMOGRAM This tracing has been studied by Lewis and Terry, [3] Boone and co workers [5] and Engstroem and co workers [14] The atrial tracing shows the absence of atrial waves The waves of the ventricular and arterial

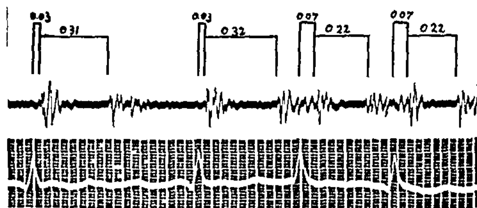


FIG 208 Ecg and phonocardiogram in a case of mitral stenosis with atrial fibrillation Variable intervals between R and the first sound

tracings (aortic and pulmonic) have a variable amplitude which is roughly proportional to the length of the previous diastole

CONCLUSIONS Atrial fibrillation is usually easily recognized by means of the electrocardiogram In cases with a rapid ventricular rate and where flutter may be suspected jugular and electrokymographic tracings may help by revealing the absence of atrial waves between the ventricular waves

Ventricular Tachycardia

Ventricular tachycardia is a serious disturbance due to the existence of an abnormal pacemaker in one of the ventricles

All factors responsible for increased excitability of the myocardium may be involved but coronary heart disease and heart failure are the most frequent causes For this reason it can be stated that ventricular tachycardia is often found in organic heart disease

ELECTROCARDIOGRAM Two main varieties have been recognized

1 *Salvos of ventricular premature beats These may number from* 10 to 100 or more and follow each other with absolute regularity

2 Real ventricular tachycardia It is similar to the first type except that the attack lasts for many hours or days (Fig 209) The beginning of the attack is sudden but it may be preceded by a few scattered premature beats having the same electrical appearance of the complexes during the attack These are broad have a high voltage are notched and are followed by a T wave which is in the opposite direction of the main complex In certain cases the P waves having a much slower rate than the ventricular complexes may be seen when they fall in diastole

Localization of the focus of origin of the attack is done as for ventricular premature beats (p 405)

After the attack is over depression of S T and inversion of T in either lead 1 or lead 3 may occur (post tachycardial syndrome) A prolongation of the Q T interval is also possible These changes appear *after* the end of the attack and may last from a few days to one month

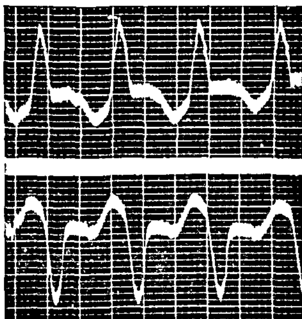


FIG 209 Ecg of ventricular tachycardia Leads 1 and 3

ARTERIAL TRACINGS The pulse waves are small and may show a variable height a higher wave is caused by the casual precedence of an atrial contraction over a ventricular There may be pulsus alternans (p 428)

JUGULAR TRACING High atrial waves are recorded at a rate which is slower than that of the arterial pulse (unless there is atrial fibrillation) The waves may be slightly irregular

PHONOCARDIOGRAM The heart sounds especially the first are loud and clear (Fig 210) [36, 4] There may be no murmurs and no triple rhythm On the other hand the atrial contraction which has a slower rate than the ventricular may cause an atrial sound either in systole or in diastole according to the casual coincidence of the two rhythms Splitting of the first sound has been *erroneously reported this splitting cannot occur because the interval separating* the contractions of the two ventricles is shorter than the duration of the main phase of the first sound

CONCLUSIONS Ventricular tachycardia is usually recognized by means of the electrocardiogram The other tracings may be used in special cases

Ventricular Flutter and Fibrillation

An abnormal mechanism of rapid excitation, similar to that more frequently observed in the atria may develop in the ventricles frequently resulting in death Animal experimentation demonstrates that ventricular flutter and fibrillation may be induced by drugs ligation of the coronary arteries injection of irritant substances into the myocardium or electric faradization of the heart Both clinical and experimental evidence indicate that impairment of the coronary circulation is the chief element to be considered

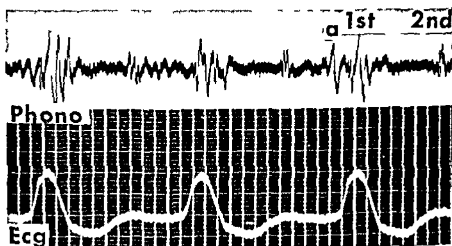


FIG 210 Phonocardiogram in a case of ventricular tachycardia *a* is a fourth sound due to an atrial contraction

ELECTROCARDIOGRAM Large regular monophasic ventricular waves follow each other at a rate of 250–300 per minute in ventricular flutter rapid irregular bizarre small complexes are typical of ventricular fibrillation

CONCLUSIONS The absence of any clinical evidence of cardiac activity is common to ventricular standstill and fibrillation The former is usually of abrupt onset and may end suddenly the latter is usually preceded by ventricular premature beats The electrocardiogram has diagnostic value

Premature Contractions (Extrasystoles)

An abnormal stimulus may arise in the heart spreading from a point which is usually outside the normal pacemaker and cause a premature contraction In order to be effective the stimulus must occur at a time when the heart muscle is not in the absolute refractory period Increased excitability may per

mit a premature beat during the relative refractory period for instance during the T wave

Premature beats may occur at any age including infancy and fetal life The mechanism of premature beats is thought to be the following

When the abnormal stimulus arises in one of the atria it spreads in all directions reaching both the s a node and the a v node By so doing it discharges the normal impulse which is forming in the s a node and may disturb the activity of the latter The pause following the premature beat is equivalent to the time interval required for the stimulus to reach the s a node plus the time between two normal contractions The pause is noncompensatory

When the abnormal stimulus arises in one of the ventricles it spreads in all directions usually reaching the a v node It is frequently prevented from reaching the atria because these are in the refractory stage or because of a protection block" The stimulus reaches the other ventricle in a roundabout way as in bundle branch block (p 429) Therefore the ventricle where the stimulus has arisen contracts first and is followed by the other

The pause following the premature beat is such that the interval between the last normal contraction before the premature beat and the first after it equals the double of a normal interval (compensatory pause) This is not true in the case of an interpolated premature beat which is actually an additional contraction

ELECTROCARDIOGRAM Several types of tracings are observed according to the rhythm and frequency of the premature beats Various types are obtained in relation to the point of origin

Atrial premature beats A normal P QRS is followed by a short diastole then by a complex which has a slightly deformed (higher broader or lower) P wave and a slightly longer or shorter P R interval the QRS is normal but may be slightly changed (aberrant) The pause following the premature beat is seldom compensatory (Fig 211) The first P wave after the premature beat may be higher and sharper than the others

Some authors admit the existence of *sinus premature beats* These are said to have a normal P wave and a normal P R and to be followed by a normal interval Slight variations in the shape of the P wave should not rule out this type of premature contraction [3]

Blocked atrial premature beats are possible but uncommon A P wave occurs near the end of a normal or premature complex it is not followed by a QRS a small flat sluggish wave may follow the P representing the terminal deflection of the atrial complex (*Ta* p 235)

When an atrial premature beat originates from a point near the a v node it is called a *nodal premature beat* Its typical characteristics are inverted or deformed P wave short P R P either preceding QRS or following it

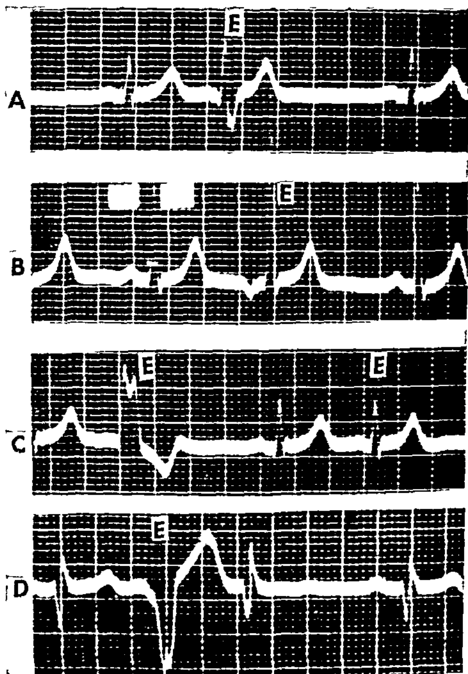


FIG 211 Ecg in cases of premature contractions (extrasystoles) *E* extrasystole *A* the second complex is an infranodal premature contraction with aberrant conduction *B* the second complex is a nodal premature contraction *C*, the first complex is a ventricular premature contraction the third is an atrial premature contraction *D* the second complex is a ventricular interpolated premature contraction

normal QRS The pause is seldom compensatory (Fig 211) Atrial premature beats are seldom interpolated

Ventricular premature beats A ventricular premature beat has the following characteristics absence of the P wave high voltage long duration and splintering of the ventricular complex, raised S T T wave frequently in the opposite direction of QRS (Fig 211)

The position of the heart causes important modifications of the direction of the complex Figure 213 shows the various patterns resulting from premature contractions occurring in the vertical average and horizontal hearts

In rare cases a ventricular premature beat may send its stimulus backwards to the atria thereby causing a retrograde atrial contraction When this

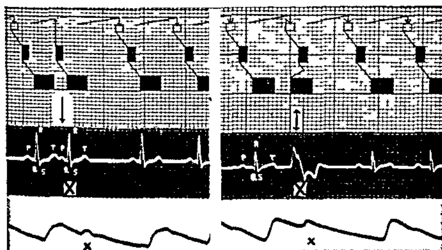


FIG 212 Scheme of atrial and ventricular premature contractions

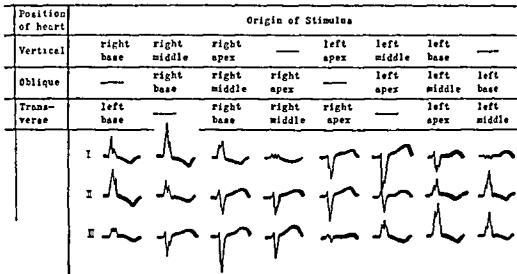
happens the pause following the premature beat is noncompensatory A ventricular premature beat may occur as an *interpolated extrasystole* This is more commonly, but not exclusively found in the slow heart (Fig 211)

A *ventricular escape* can be easily differentiated from a premature beat because it occurs after a long pause has a normal ventricular complex is not preceded by an atrial wave and is not followed by a compensatory pause

LOW FREQUENCY TRACING CARDIOGRAM The premature contraction may give waves of a normal configuration The prolonged tension period causes sometimes bizarre or large waves

JUGULAR TRACING Atrial premature beats have *a* *c* and *v* waves which are similar to those of a normal tracing Nodal and ventricular premature beats on the other hand frequently have as high a wave

A left ventricular premature beat may show a delay of *v* over the second sound complex (earlier contraction of the left ventricle)[10]



Position of heart	Origin of Stimulus							
Vertical	right base	right middle	right apex	—	left apex	left middle	left base	—
Oblique	—	right base	right middle	right apex	—	left apex	left middle	left base
Transverse	left base	—	right base	right middle	right apex	—	left apex	left middle

FIG 213 Ventricular premature contractions in the various positions of the heart (after Holzmann)

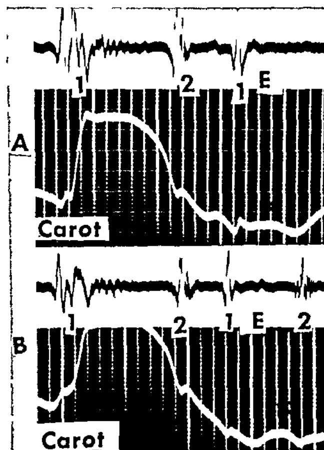


FIG 214 Phonocardiograms and carotid tracings of premature contractions ($E\ E$) A, the premature contraction produces only a first sound B the premature contraction produces two sounds x x Small pulse of the premature contraction

PHONOCARDIOGRAM Several entirely different phenomena can be recorded The first sound is usually normal or louder than the others (Figs 214 and 215) The beginning of the sound is delayed over the beginning of QRS in a ventricular premature beat (Fig 215) The two valvular phases of the first sound may be widely separated on account of the prolongation of the tension period and this may simulate splitting of the sound (Fig 215 B) The second sound of the premature beat is normal or weaker than the others and may be absent (Fig 214) It is frequently split [10]

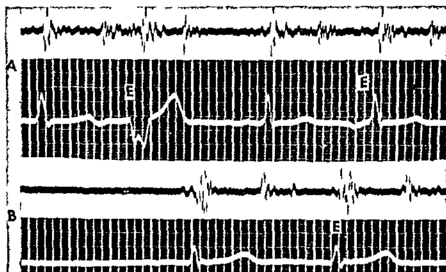


FIG 215 Phonocardiograms of premature contractions
A The second complex (*E*) is a ventricular premature contraction long delay between beginning of ecg complex and beginning of first sound loud second sound The fourth complex is a nodal premature contraction prolonged first sound
B The second complex (*E*) is an atrial premature contraction The first sound has a wide separation of the two valvular components and seems split

A premature beat may be followed by either a diastolic murmur or a diastolic sound which do not occur after normal contractions

ARTERIAL TRACING HEMODYNAMICS The pulse wave of the premature beat may be as high as a normal wave and occasionally higher It is usually smaller and may be absent (Fig 216) Records taken over different arteries show that the pulse wave of the premature beat may be present in the arteries near the aorta and absent in those distant [30] A drop in pressure occurs during the compensatory pause The following wave is higher than normal and is followed by a smaller wave this may start a pulsus alternans

The pulse of the premature contraction has a low systolic and a high diastolic pressure while the following pulse is much larger on account of high systolic and low diastolic pressure From two to three pulse waves are necessary for reaching again a normal and stable level of blood pressure [1] On the other hand, the first normal contraction following an interpolated premature beat frequently yields a smaller pulse wave in the peripheral arteries [29]

The pulse wave of a right ventricular premature beat may be delayed over the beginning of the corresponding first sound (earlier contraction of the right ventricle) [8]

The stroke volume varies greatly being inversely proportional to the length of the preceding diastole Therefore, the earlier the contraction the smaller the stroke volume The compensatory pause is followed by a con

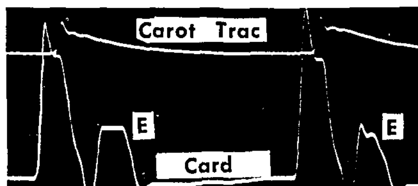


Fig 216 The arterial pulse of a premature contraction may be absent If there is bigeminy, the pulse simulates that of bradycardia

traction with proportionally larger stroke volume if the myocardium is normal [1]

The speed of the pulse wave is directly proportional to the level of diastolic pressure therefore the weak pulse of the premature contraction has a greater velocity than the others [1]

Blood Pressure Tracings Different levels of blood pressure can be obtained if the premature beats occur periodically (1) those corresponding to the systolic and diastolic pressure of the normal pulse waves (2) those corresponding to the pulse waves of the premature beats (low systolic, high diastolic) and (3) those corresponding to the pulse waves immediately subsequent to the premature beats (high systolic, low diastolic)

Electrokymogram Premature contractions are easily studied by means of border tracings of the left ventricle the pulmonary artery or the aortic arch If simultaneous tracings of the left ventricle and one of the arteries (aorta or pulmonary artery) are taken conclusions may be drawn about the site of origin of a ventricular premature beat The aspect of the tracing is dependent

upon the time of the cardiac cycle at which the premature contraction occurs and the phase of ventricular filling [14]

If the contraction starts early in diastole its amplitude is small isometric contraction is prolonged and the curve assumes a more peaked aspect The prolongation of tension time due both to the high level of arterial pressure and the small amount of ventricular blood may last more than 0 06 second [14] If the contraction takes place immediately after the T wave no pulsations of the aorta can be recorded During the compensatory pause an abnormally large filling of the ventricles takes place This plus the lower level of arterial pressure causes a shortening of the isometric contraction period of the following contraction

CONCLUSIONS Diagnosis of premature contractions is usually made by electrocardiography The other tracings are useful in order to gather supporting evidence for explanations of diastolic sounds or murmurs connected with the arrhythmia and for the study of the peripheral disturbance caused by the arrhythmia

Escapes

Escapes (or escaped beats) occur whenever the activity of the a v node or one of the ventricles reveals itself during a long pause they are therefore due to a phenomenon of default Escapes may occur in the following conditions (1) in pronounced sinus arrhythmia (2) in partial a v block or atrial fibrillation (3) in cases with ventricular premature beats (4) during standstill of the heart caused by strong vagal stimulation (compression of the eyeballs) when both the s a node and the a v node are inhibited

Escapes are recognized in the electrocardiogram by the normal configuration of the ventricular complex

ABNORMAL RHYTHMS 2 DISTURBANCES OF RATE AND RHYTHM DUE TO DECREASED CONDUCTIVITY

Nodal Rhythm

Nodal rhythm is a regular and slow rhythm of the heart set up by impulses from the a v node The activity of this node becomes apparent in three conditions

1 When an interruption of the conducting system prevents the stimuli of the normal pacemaker from reaching the a v node In such cases the atria are controlled by the s a node and the ventricles by the a v node there is a *double rhythm*

2 When there is atrial fibrillation or atrial standstill * In such cases the atria are immobile and ventricular contraction is controlled by the a v node

* Atrial standstill or atrial paralysis is not commonly admitted However well studied cases prove its existence

3 *When the activity of the s a node is suspended or depressed* This is the typical a v nodal rhythm and occurs mostly in aged people

The disappearance of the sinus rhythm is not felt by the patients, as the a v node takes over the function of the pacemaker However, the new center is less efficient because its rate is slower the normal succession of atrial and ventricular contractions is not preserved and the influence of the cardiac nerves on the new pacemaker is nearly absent

In typical nodal rhythm the atria are stimulated in a retrograde direction by the a v node If the stimulus starts from the upper part of the node the atrial contraction slightly precedes the ventricular If, on the other hand the stimulus starts from the lower part of the node, the ventricles contract first

In all the above mentioned conditions there is a "nodal rhythm by default , only the lack of a descending impulse enables the a v node to take over the pacemaking function A different type of nodal rhythm occurs when there is such an increase of a v node excitability that this center becomes the pace maker even when the s a node is normal This form has been called nodal rhythm by usurpation [2] and has been already described (p 393)

ELECTROCARDIOGRAM The electrocardiogram reveals a series of normal ventricular complexes The P wave is usually inverted and very near QRS a fact which is most apparent in leads 2 aVR and aVL The P wave may precede or follow QRS by about 0 05 second Unusual course of the stimulus in the atrial walls (or coincidence of two independent rhythms) may cause the less common occurrence of an upright P wave If the atrial contraction follows the ventricular by more than 0 2 second a second ventricular con traction may follow producing a bigeminal rhythm with one atrial wave between the two ventricular waves

PHONOCARDIOGRAM The phonocardiogram shows that the atrial sound is missing and the first sound is prolonged

JUGULAR TRACING The jugular tracing may show fusion of the *a* with the *c* wave the new wave is high

Sino Atrial Block

Sino atrial block may be incomplete or complete Incomplete s a block may be based on (1) prolongation of the s a conduction time, (2) progressive *increase of s a conduction followed by absence of a complete atrioventricular* cycle or (3) sudden absence of an atrioventricular cycle

Complete s a block may cause *diastolic standstill* of the whole heart or shift of the pacemaker to a lower center (nodal rhythm p 409)

ELECTROCARDIOGRAM *Occasional s a block* A regular series of normal P QRS complexes is followed occasionally by a long diastolic pause The length of the cycle including this pause is twice that of either the preceding or the following cycle If this pause occurs regularly, there is a special type of periodic s a block

Periodic s a block The P QRS complexes are gradually more and more distant being separated by gradually increasing diastolic pauses After a certain number of them a short diastole follows the length of the short interval between Q and the following Q is equivalent to one half of that of the immediately preceding interval and represents the only normal distance on which measurement should be based

If the block occurs every second beat the ecg simulates that of sinus brady cardia because no wave reveals the sinus impulses and the rate of the atria and ventricles is from 30 to 40 The disturbance is revealed by the fact that either spontaneously or after atropine the rate suddenly doubles (from 40 to 80 from 35 to 70)

In certain tracings of s a block a second rhythm occurs during the long pause (*double rhythm type Rothberger*[49]) this is revealed by complexes of a different shape following each other at a more rapid rate

ARTERIAL TRACINGS In occasional block the tracings reveal the sporadic absence of a pulse wave (intermittence)

In periodic block the series of pulse waves shows a periodic increase of the interval separating two pulse waves After a certain number there is a longer pause where diastolic pressure drops more

Atrioventricular Block

Impaired conduction (block) of the stimulus between the s a node and the a v node within the a v node or in the bundle of His is the cause of this dis turbance

When the conducting tissues fail to transmit the excitation from the s a node to the ventricles a new pacemaker takes over the automatic functions this may be located in the a v node (nodal rhythm) in the bundle of His (septal rhythm), or in one of the ventricles (idioventricular rhythm)

The block may be caused by (1) a congenital lesion (2) an inflammatory or degenerative lesion (3) metastatic nodules (4) areas of fibrosis (coronary heart disease) or (5) a functional or vascular disturbance of the conducting tissues

ELECTROCARDIOGRAM There are different types of tracings

1 *Latent a v block* revealed by prolongation of the P R interval up to 0 30 second (so called Grade 1 a v block)

2 *Occasional a v block* Sporadically a P wave is not followed by a ventricular complex

3 *Periodic a v block* (type I of Wenckebach)[65] The P waves have a regular succession, the P R intervals gradually become longer until one P wave is not followed by a ventricular complex (blocked P) The entire cycle from one normal P QRS to the following normal is called the Luciani Wenckebach period (from the names of the authors who described it in animals and man) (Fig 217 A)

A curious type of tracing occurs at times in partial block, it has been called *interference dissociation* [2, 40] The P and QRS waves have similar rates, but no fixed relationship, so that P may precede or follow QRS From time to time a seemingly normal P QRS takes place through casual coincidence of the waves

4 *Intermittent a v block* (Type II of Wenckebach,[55] also known as *Mobitz type*[41] *of a v block*) The number of atrial complexes is a multiple of the ventricular such as 2 to 1, or 3 to 1 In the 2 to 1 cases there is a blocked P a normal P QRS then another blocked P

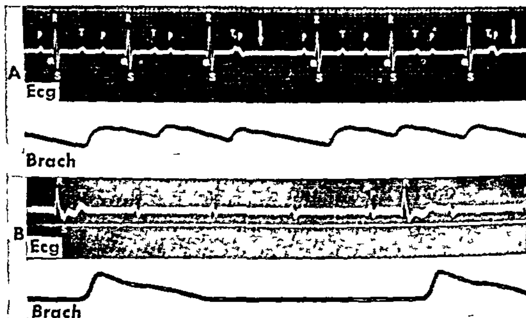


FIG 217 A V block Ecg and pulse *A* Type I of incomplete a v block (periodic a v block) *B* complete a v block

5 *Complete a v block* While types 2 to 4 are also called *partial a v block* in other cases the block may be complete The atrial waves have a normal rate (60 to 90) while the ventricular have a slow rate (15 to 40) and there is no relationship between the two Two to five P waves may be present in the intervals between the QRS while some of them are superimposed on or masked by the ventricular complex (Fig 217 B) The blocked P waves often are followed by an after deflection called the T atrial wave (*Ta wave*) which has the same meaning as the T wave of the ventricular complex (p 235) In *congenital block* the rate of the ventricles may be faster (from 40 to 60) [9]

It is commonly stated that bundle branch block frequently complicates a v block " Actually the idioventricular rhythm arises in one of the branches of the bundle of His so that the stimulus spreads first to the ipsilateral then to

the contralateral ventricle The ventricular complexes resemble those of bundle branch block or paroxysmal ventricular tachycardia There is some resemblance with ventricular tachycardia except that here the phenomenon is caused by default and that the rate is slow

A *Stokes Adams attack* is preceded by either increase or decrease of the atrial rate During the attack the ventricular rate may slow down further On the other hand there may be ventricular standstill ventricular tachycardia or ventricular flutter

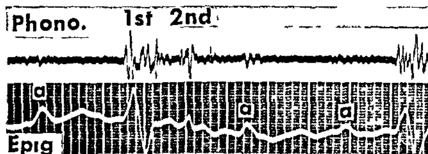


FIG 218 Complete a v block Atrial sounds and atrial pulses (*a a*), recorded between the ventricular contractions Phonocardiogram and epigastric tracing

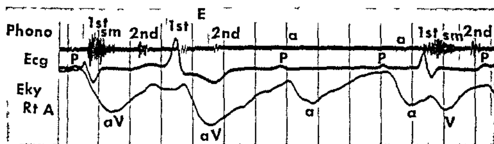


FIG 219 Phonocardiogram and electrokymogram of the right atrial border in a case of complete a v block with premature ventricular contractions

If the atrial rate is very slow and that of the ventricles is relatively rapid the number of P waves becomes equivalent to that of the QRS complexes Small independent changes in rate of the two pacemakers modify the lengths of the P R intervals This singular phenomenon is called *isorhythmic dissocia tion*

PHONOCARDIOGRAM Atrial sounds may be present during diastole In general one dull sound follows each P wave (Figs 218 219) In exceptional cases an atrial contraction is followed by two sounds [13, 28, 36] the second may be due to a vibration of the a v valves [13] In an unusual case three groups of vibrations

followed each P wave [8] These were explained as due to the atrial contraction the distention of the ventricular wall and the elastic reaction of the latter The atrial sound is louder in children, old people, and persons with a flat chest The vibrations of an apical systolic murmur are frequently recorded, those of blowing apical diastolic murmurs coincident with the atrial contractions are recorded at times in elderly patients with a calcified mitral valve [50] In cases of latent block the first sound complex is of lower intensity [60] When atria and ventricles contract at the same time, a louder first sound is recorded (*cannon sound*)

Jugular Tracing The *a* waves are normal in shape and rate, the *c* and *v* waves occur at regular but much longer, intervals Therefore, more than one *a* wave is found between a *v* wave and the next *c* wave Occasional coincidence of an *a* wave with a *c* or *v* may occur with a resulting higher wave

Cardiogram High atrial waves are present between the undulations caused by the ventricular contractions

Arterial Tracing A high and slow pulse is recorded An anacrotic depression is frequently present in the ascending branch of the curve Multiple undulations follow the dicrotic wave

In occasional block the tracings reveal the occasional absence of a pulse wave (intermittence) In periodic block, the series of pulse waves shows a periodic increase in the pause separating two pulses, then a longer pause with a deeper pressure drop Interference dissociation may cause a bigeminal rhythm the second wave being paradoxically higher than the first [15]

Electrokymography The electrokymogram of a v block has been studied by the author,[33 35] Engstroem and coworkers,[14] and Pannier and coworkers [43]

Atrial tracings If atrial contraction falls during the ventricular, the atria decrease in volume as in normal subjects apparently the blood is pushed backwards into the veins If the atrial contraction falls during ventricular diastole, the pattern consists of an oblique descending line during atrial contraction and a less steep rise subsequent to it The depth of the contraction varies it is usually larger when the atrial contraction falls during a ventricular contraction because of additional ventricular pull (Fig 219)

Left ventricular tracing The tracing of the left ventricle may not show any effect of the atrial contractions If it does the latter are revealed by small positive waves

Conclusions Complete a v block is usually recognized by simple palpation of the pulse Incomplete block may be more difficult to diagnose clinically while diagnosis is easily made by the electrocardiograph The other tracings (phonocardiogram electrokymogram venous and arterial tracings) give supportive evidence and are used mostly for an accurate study of the patient

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CHAPTER 48

Diffuse Myocardial Lesions

CARDIAC HYPERTROPHY AND STRAIN

Cardiac enlargement is a common finding in heart disease and may be due to one of the following causes

1 Valvular lesions or malformations placing strain on one, two or more cardiac chambers (rheumatic or bacterial valvular lesions congenital heart disease)

2 Persistent increase of resistance in the vessels of either the greater or the lesser circulation placing strain on one of the ventricles (hypertensive heart disease, pulmonary heart disease)

3 Diffuse structural lesion or functional disturbance of the cardiac muscle (inflammation or degeneration metabolic or endocrine disturbance, vitamin deficiency)

4 Diffuse lesion of the coronary system causing impairment of the blood supply to the heart (coronary heart disease)

In the above mentioned conditions cardiac enlargement is due to dilatation hypertrophy, or both

Hypertrophy of the heart muscle is not accompanied by proportional development of its capillary network [37] This ultimately leads to some degree of ischemia, especially in the walls of the left ventricle where intramural flow is opposed to a greater extent by systolic increase of pressure Therefore an electrocardiographic picture once called "strain pattern, and actually caused

by ischemia, is frequently encountered in ventricular hypertrophy This may be followed by that of intraventricular block or bundle branch block because ischemia leads to fibrosis and fibrosis may prevent spreading of the stimulus in certain parts of the conduction system

Electrocardiogram

General data indicating ventricular hypertrophy are [1 1 15 38 39]

1 High voltage in the limb leads (unipolar and standard) due to increased thickness of the ventricular wall

2 Marked axis deviation due to increased surface and thickness of one ventricle

3 Increased duration of QRS (0 10–0 11 second) because longer time is required for the stimulus to reach the epicardial surface of the thickened ventricular wall

4 Delay between beginning of the complex and intrinsicoid deflection in the chest leads of the hypertrophied side due to the same mechanism

5 Secondary changes of S T and T Following an altered course of depolarization ventricular repolarization of the hypertrophied ventricle begins after that of the normal ventricle However measurements of the ventricular gradient indicate that there may also be primary changes of T due to relative ischemia

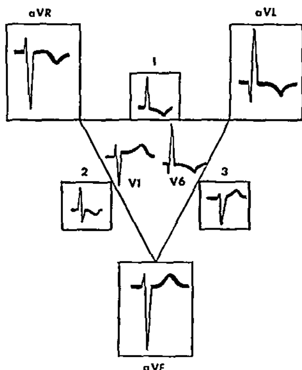


FIG 220 Electrocardiographic pattern of left ventricular hypertrophy and strain horizontal position and counter-clockwise rotation of the heart

Left ventricular hypertrophy is usually accompanied by a marked left axis deviation * this is easily evaluated by the White Bock index which is above +17 mm [3] † If there is hypertrophy of the septum there may be a deep Q wave in the chest leads (Fig 220) [5]

* This deviation is absent in the vertical heart
† The White Bock index is found by using the formula $(R1+S3)-(S1+R3)$

The ecg details vary with the position of the heart and are summarized in Tables 22 to 24

TABLE 22 LEFT VENTRICULAR HYPERTROPHY WITH VERTICAL HEART*

Lead	*Pattern of ecg*
V1–V4	rS slightly elevated ST segment upright T wave
V5–V6	qR depressed ST segment inverted T wave slight delay of the intrinsicoid deflection
aVR	QS or rS deflection with diphasic or inverted T wave
aVL	QS or rS and upright T wave
aVF	qR with depressed ST segment and downward T

* In these cases the standard leads may show right axis deviation with depressed ST and downward T wave in 2 and 3

TABLE 23 LEFT VENTRICULAR HYPERTROPHY WITH HORIZONTAL POSITION AND COUNTERCLOCKWISE ROTATION OF THE HEART

Lead	*Pattern of ecg*
V1–V2	rS or QS slightly elevated ST segment upright T
V3–V6	qR depressed ST segment downward T wave slight delay of the intrinsicoid deflection
aVR	rS or AS downward T wave
aVL	qR depressed ST segment downward T wave
aVF	rS upright T wave

TABLE 24 LEFT VENTRICULAR HYPERTROPHY WITH HORIZONTAL POSITION AND MARKED CLOCKWISE ROTATION OF THE HEART

Lead	*Pattern of ecg*
V1–V6	rS deflection slightly elevated ST segment upright T wave
aVR	QR downward T wave
aVL	Qr depressed ST segment downward T wave
aVF	rS upright T wave

The pattern of *right ventricular hypertrophy* is not easily schematized because it may be caused by various clinical conditions Several pictures have been described [36]

1 Normal electrocardiogram due to the predominance of the left ventricle in spite of a moderate right ventricular hypertrophy

2 There is right axis deviation revealed by the fact that the White Bock index (p 419, footnote) is less than −14 R has a high voltage in V1 V2 (7 mm or more) while S has a high voltage in V5 V6 (7 mm or more) Thus the ratio $\frac{(R\ S)\ \text{in V5}}{(R\ S)\ \text{in V1}} = 0\ 4$ or less There may be evidence of associated right atrial hypertrophy (Fig 222)

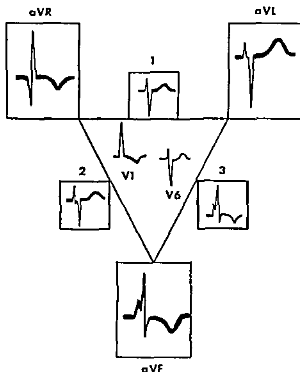


FIG 221 Average electrocardiographic pattern of right ventricular hypertrophy and strain

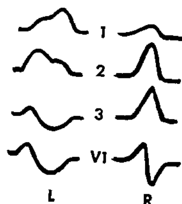


FIG 222 Electrocardiographic patterns of left (*L*) and right (*R*) atrial hypertrophy

3 An average picture of right ventricular hypertrophy is summarized in Table 25 and is schematized in Fig 221

TABLE 25 MARKED RIGHT VENTRICULAR HYPERTROPHY

Lead	*Pattern of ecg*
V4R V1 (possibly 2)	R or Rs depressed ST segment inverted T wave
V5–V6	RS normal ST segment upright T wave
aVR	QR or qR inverted T wave
aVL	rS upright T wave or QR *inverted* T wave
aVF	rS or QR *inverted T wave*

4 There is an additional *focal block* of the right side (pp 430 and 437) The right chest leads have an rSR or rsR pattern and an inverted T There are no further changes of the limb leads due to the focal block

Atrial hypertrophy is revealed by changes of the axis of P as well as by changes of its duration and configuration Two patterns have been recognized[35] (Fig 222)

1 Pattern of *right atrial hypertrophy* encountered in chronic cor pul

monale and in congenital heart disease There is high voltage of P in 2, 3, and aVF while V1 and V2 present a P wave made of a high positive phase followed by a brief and pointed negative phase

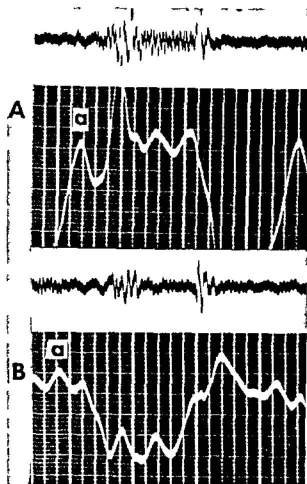


FIG 223 Low frequency tracings in a case *with left ventricular hypertrophy (hypertensive* heart disease) *A* Large, plateau like impact at apex *B* negative thrust at epigastrium Both tracings have a high atrial wave (*a a*) but this is larger over the apex Superimposed tracings

2 Pattern of *left atrial hypertrophy* encountered in mitral valve lesions There is a notched P in 1 and aVL and a diphasic P with deep and broad negative phase, in 3 and aVF V1 and V2 present a P wave having a deep and broad negative phase

In both patterns, P lasts more than 0 10 second

Low Frequency Tracings

Enlargement of one of the ventricles may be revealed by low frequency tracings [10]

1 Left ventricular hypertrophy is revealed by a high often plateau like positive thrust *at the apex*, at the same time the epigastric tracing has a negative thrust (Fig 223)

2 Right ventricular hypertrophy is revealed by a high, early systolic thrust *at the epigastrium* while the apical tracing has a negative thrust (*Fig 224*)

3 A *large atrial wave* may be observed in either tracing and is evidence of increased atrial pressure

TRIPLE AND QUADRUPLE RHYTHMS OF THE HEART

The names triple rhythm and quadruple rhythm have been suggested[10] to indicate those cadences which are due to addition of one or two diastolic sounds to the more commonly heard two heart sounds Older and well known

names were *gallop rhythm*[5] and *train wheel rhythm*[17] The new definition is more comprehensive and includes several possibilities which were not considered in the older ones

As already mentioned (pp 14 and 41) the normal heart may have four sounds The two loudest are the first and second sounds at the beginning and end of ventricular systole (*systolic sounds*) the other two of less intensity occur during ventricular diastole in coincidence with the phases of rapid filling of the ventricles They are the third sound and the fourth or atrial sound (*diastolic sounds*) They are usually inaudible in adults with the exceptions of persons with a flat thin chest who may have an audible third sound

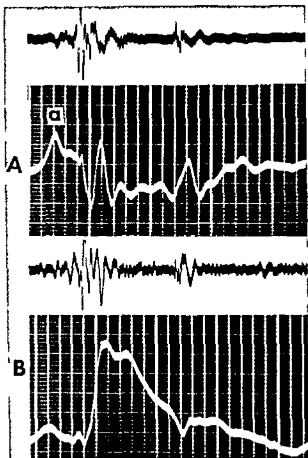


Fig 224 Low frequency tracings in a case with right ventricular hypertrophy (chronic cor pulmonale) *A* Mostly negative thrust at apex High atrial wave (*a*) *B* Large positive impact in early systole at epigastrium Superimposed tracings

Until recently a *triple rhythm* (gallop) was considered as a purely auditory phenomenon[16] However phonocardiography has revealed that in certain cases with a triple rhythm the diastolic sound is inaudible because it is weak low pitched or near the first sound[9] The author with Roitman tried to separate the cases with a more physiologic triple rhythm from those with a more pathologic triple rhythm It was suggested to consider more pathological any case having a diastolic sound with the following graphic characteristics (1) frequency of vibrations of 30 or more per second irrespective of its amplitude (2) amplitude greater than two thirds of the loudest of the two main sounds at the apex or (3) both

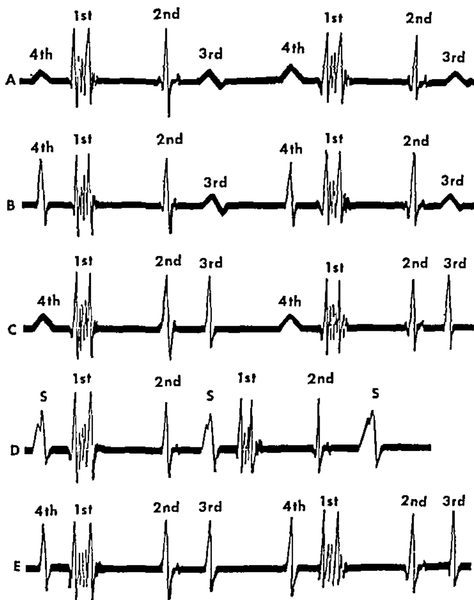


FIG 225 Phonocardiographic scheme of the most common types of triple and quadruple rhythms (gallop or train wheel rhythms) *A*, Normal sounds *B* atrial type of triple rhythm (loud fourth sound) *C*, ventricular type of triple rhythm (loud third sound) *D* summation type of triple rhythm (fusion of loud third and fourth sounds) *E*, quadruple rhythm (loud and separated third and fourth sounds)

It should be noted that this empirical classification has no absolute value because the pathologic is merely the accentuation of a physiologic phenomenon

PHONOCARDIOGRAM LOW FREQUENCY TRACING Three types of triple rhythm and one of quadruple rhythm have been recognized (Fig 225)

1 *Atrial type* The additional sound occurs at the time of atrial contraction and represents the accentuation of the fourth sound (Fig 226 A) It usually falls in late diastole but if the P R interval is long and diastole is short it may fall in early diastole

2 *Ventricular type* The additional sound falls in early diastole at the time of rapid filling and represents the accentuation of the third sound It occurs from 0 14 to 0 18 second after the second sound (Fig 226 B)

3 *Summation (or nondescript) type* There is an additional sound or a short rumbling murmur in mid diastole This is due to more or less simultaneous occurrence of rapid passive filling and atrial contraction causing the rapid succession or fusion of the third with the fourth sound (Fig 226 C)

4 *Quadruple rhythm* There are two additional sounds in diastole representing the accentuation of the third and fourth sound (Fig 227)

The diastolic sound is made of a high diphasic or triphasic

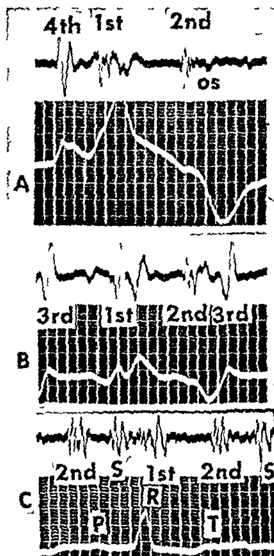


FIG 226 Phonocardiograms of three cases with triple rhythms *A* atrial type due to increased loudness of the fourth sound (stethoscopic and linear tracings) *B* ventricular type due to increased loudness of the third sound (stethoscopic and linear tracings), C summation type where a complex sound (*S*) exists in diastole Stethoscopic tracing and electrocardiogram

vibration It is simultaneous with a high wave in the low frequency tracing (cardiogram or epigastric tracing) [19] In the summation type, two distinct waves may be present in the low frequency tracing even if a prolonged sound or short rumble is revealed by the sound tracing

Phonocardiograms recorded first over the apex and then at the epigastrium reveal whether the triple rhythm originates in the left or in the right ventricle A triple rhythm recorded only at the apex usually reveals left ventricular strain, a triple rhythm recorded only at the epigastrium usually reveals right ventricular strain Other types of triple rhythms are recorded over both areas

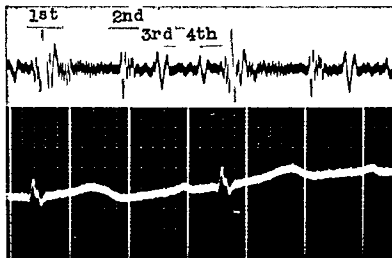


FIG 227 Phonocardiogram in a case with a quadruple rhythm

The following conditions increase the loudness of the diastolic sounds

1 *Rapid heart action* The short diastole causes a tumultuous filling of the ventricles

2 *Prolonged interval between atrial and ventricular contractions* the increased interval permits a better audition but does not change the graphic characteristics of the atrial sound

3 *Hypertrophy of the left atrium* as observed in mitral insufficiency without severe mitral stenosis gives a powerful contraction displacing a large amount of blood The same is true for the *right atrium* in cor pulmonale or mitral stenosis This increases the loudness of the fourth sound

4 *Dilated and weak myocardium* the ventricular wall vibrates more when hit by the fluid wave This is common in congestive failure and in coronary heart disease

5 *Ventricular strain* favors triple rhythms through increase of atrial pres sure this is typical of hypertensive heart disease

A diastolic type of triple rhythm* is recognized by recording simultaneously a stethoscopic phonocardiogram and a low frequency tracing (apex cardio gram or epigastric tracing) The atrial type is also recognized by comparison with an electrocardiogram the additional sound precedes the QRS complex

Differential diagnosis between splitting of the second sound opening sound or opening snap of the mitral valve and a diastolic sound, apart from the time coincidence with the waves of the low frequency tracing (cardiogram) and the electrocardiogram may also make use of the following average fig ures (adults)

1 The distance between the two phases of a split second sound varies be tween 0 025 and 0 05 second [3] (older data[40] were 0 03–0 11 second but the last figure should not be accepted without better proof)

2 The distance between the main phase of the second sound and the physiologic opening sound of the mitral valve varies between 0 04 and 0 07 second [3]

3 The distance between the main phase of the second sound and the path ologic opening snap of the mitral valve varies between 0 07 and 0 11 second [3] (0 07–0 13 in older studies) [40]

4 The distance between the main phase of the second sound and the beginning of the third sound is between 0 12 and 0 18 second [21, 3, 27]

A dull *third sound* is normal in children or young persons with a thin flat chest It is abnormal in older people or if very loud [3] A *fourth sound* may be recorded (seldom heard) in normal subjects above the age of 40 It becomes an abnormal phenomenon if it is found in younger people or if the vibration is large or high pitched [23]

As already stated triple and quadruple rhythms are commonly associated with tachycardia ventricular dilatation or ventricular strain Therefore these rhythms are frequently encountered in pregnancy thyrotoxicosis fever anemia rheumatic or congenital heart diseases and hypertensive or coronary heart diseases The rhythm is caused by functional phenomena which may disappear (delivery sedation digitalization sympathectomy or venesection) causing a reversal to normal auditory phenomena

While a triple rhythm is more frequent in cardiac patients than in normal subjects it may be found occasionally in the latter because its production is favored by functional elements which may be present without heart disease

The term triple rhythm should be preferred to others in order to dispel the unjustified fears once attached to the term gallop rhythm However it should be kept in mind that a triple rhythm is evidence of a functional dis turbance of the heart

* Addition of a sound in systole causes a systolic type of triple rhythm (p 461)

Conclusions

Recognition of a triple or quadruple rhythm is based on the phonocardio gram recorded first with an electrocardiogram and later, with a low frequency tracing of the apex or epigastrium

PULSUS ALTERNANS

Pulsus alternans occurs when every other pulse wave is weak although there is no irregularity of the heart (Fig 228)

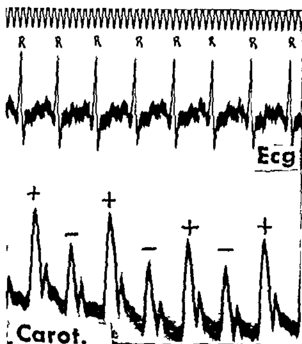


Fig 228 Pulsus alternans

Pulsus alternans may appear during attacks of paroxysmal tachycardia It is typical of hypertension, coronary heart disease (especially after myocardial infarction), and congestive failure

Pulsus alternans is recognized by palpation of the radial pulse only when exceptionally severe Manual compression of the brachial artery may cause halving of the pulse and reveal less marked alternation

Blood pressure tracings easily reveal the alternans If the patient has a heart rate of 60 decompression first shows the sounds due to the large waves (rate of 30) then those due to all waves (rate of 60) A new halving of the rate may be observed near the level of diastolic pressure

There are four types of alternating pulse [7, 26] (1) alternation of the peak (systolic alternation), (2) alternation of the foot (diastolic alternation) (3) alternation of both (systolic and diastolic alternation) (4) alternation of the dicrotic wave In exceptional cases the tibial tracing fails to reveal an alternation which is present in the brachial tracing

Several phenomena may alternate in cases of pulsus alternans

1 The intensity of the cardiac sounds (Fig 229)
2 The intensity of a murmur
3 The amplitude of the ventricular contractions as recorded by the electrokymograph

At times the electrocardiogram reveals an alternation of P R T or S T which may be concordant or discordant in relation to the pulse

Exertion usually increases the alternation A premature beat may start the alternation which usually continues for some time thereafter

Differential diagnosis from a bigeminal pulse is made by observing an electrocardiogram and a pulse tracing The ventricular complexes are identical in pulsus alternans while the unusual complex of a premature contraction periodically follows a normal complex in bigeminal pulse The small wave is nearer the preceding large wave in bigeminal pulse

In certain cases with rapid respiration the cardiac rate may be only twice the respiratory rate with secondary alternation of the pulse Inviting the patient to hold his breath for a few seconds reestablishes equal pulses

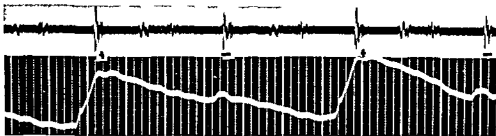


FIG 229 Alternation of the second heart sound (aortic area) and of the pulse (brachial)

The pulsus alternans of paroxysmal tachycardia and that occurring for only five or six contractions after a premature beat are of limited importance Nevertheless they add severity to the syndrome The pulsus alternans of all other conditions has a serious prognosis because it is evidence of severe left ventricular damage

Conclusions

Graphic diagnosis of pulsus alternans is simple because this is a regular pulse with alternately smaller and larger waves

BUNDLE BRANCH BLOCK

Impairment in the transmission of the stimulus is not limited to the bundle of His (p 9) If it occurs below the bifurcation of the bundle it may delay or block the conduction toward one ventricle When the delayed conduction is in one of the stems of the bundle there is *bundle branch block* (b b b) When there is diffuse impairment in the more distal subdivisions there is an

intraventricular block (i v block) When there is localized impairment in one of the distal branches, there is a *focal block*

These disturbances fail to cause any apparent change of the heart action because the affected ventricle is stimulated in a devious way through the septum On the other hand this process causes delay of stimulation and contraction of one ventricle in comparison with the other the non simultaneous occurrence of electrical and mechanical events may cause changes of the electrocardiogram electrokymogram, phonocardiogram, and mechanical tracings

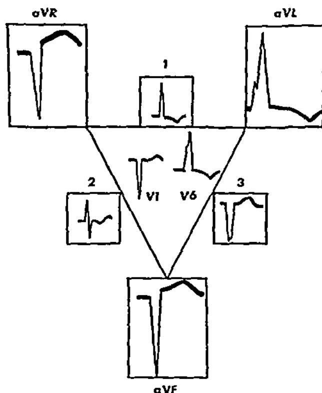


Fig 230 Electrocardiographic scheme of left bundle branch block in a horizontal heart

Electrocardiogram

The electrocardiogram recorded in the limb leads shows that QRS is broader and lasts more than 0 12 second in adults (more than 0 10 second in children) and is notched or slurred

Left Bundle Branch Block There is no S wave in lead 1 * The T wave may be in either the same or the opposite direction of the main QRS complex [13] The direction of the main wave in leads 1 and 3 fails to give a clue it may be of either the concordant type (right axis deviation) or the discordant type (left axis deviation)[4, 35] according to the position of the heart

The precordial leads give basic data there is a small R wave followed by a deep and broad S (rS) in leads V1 and V2 on the other hand the R wave complex in leads V5 and V6 (rR or M) (Fig 230), there is no Q wave in V5 V6 *because the septum is stimulated from right to left*

The data supplied by the unipolar limb and precordial leads[35] are summarized in Table 26

* A small and broad S1 (and S in V6) can be observed in cases of intraventricular block of the left base (S-type of left i v block)

TABLE 26 ELECTROCARDIOGRAPHIC CHANGES IN LEFT BUNDLE BRANCH BLOCK

Position of the heart	*Unipolar lead*	*Pattern of electrocardiogram*
Vertical heart (less common)	Rt arm	QS raised S-T upright T
	Lt arm	QS or rS small upright T
	Lt leg	R depressed S-T inverted T
	V1-V4	QS or rS upward T
	V5-V6	R depressed S-T inverted T
Horizontal heart	Rt arm	QS upward T
	Lt arm	R inverted T
	Lt leg	QS upward T
	V1	QS upward T
	V2-V3	rS upward T
	V5-V6	rR inverted T

The intracardiac electrocardiogram (right ventricle) presents a deep QS complex a positive T wave and an upward displacement of S T[34]

In left ventricular hypertrophy, leads facing the epicardial surface of the left ventricle have a wide qR pattern In left bundle branch block leads facing the epicardial surface of the left ventricle show a wide R and no Q

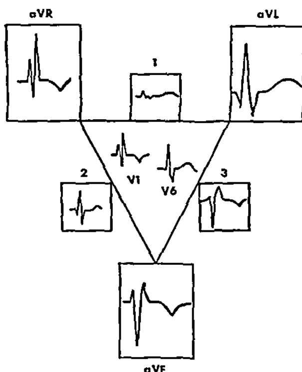


Fig 231 Electrocardiographic scheme of right bundle branch block in a horizontal heart

Right Bundle Branch Block The T wave is usually in the opposite direction of the main wave of QRS The main wave in leads 1 and 3 is usually of the concordant type (right axis deviation) However if the heart is vertical it is possible to observe a discordant type of electrocardiogram and no S wave

The precordial leads give basic data the R wave is of great amplitude and is delayed 0 06–0 08 second over the beginning of the complex in lead V1 and V2 (like rSR or M) it is of high voltage and occurs early in leads V5 and V6 (like qRS or RS) (Fig 231)

A special variety of right bundle branch block is called *Wilson type of block*. Lead 1 has a tall R followed by a deep and wide S, and by an upright T wave. Lead 3 has a qR type of complex and an inverted T (Fig 232). The chest leads V5 V6 have a slurred, wide S and R′ wave.

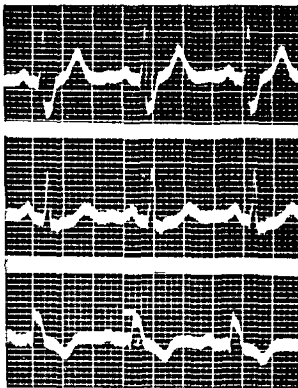


FIG 232 Wilson type of right bundle branch block

The data supplied by the unipolar limb and precordial leads[35] are summarized in Table 27.

The intracardiac electrocardiogram (right ventricle) presents a diphasic initial complex of the RS type followed by an inverted T wave.

Phonocardiogram

The first sound is prolonged and lasts more than 0.16 second. It is frequently of low amplitude. Splitting of this sound has been described by several authors.[3, 4, 41] However this observation is not confirmed by stethoscopic tracings[9] and should be attributed to inadequate technic. Ventricular asynchronism results in the delay of contraction of one ventricle of about 0.04–0.05 second. This is too short for causing a splitting of that central phase of the first sound which is clearly visible and which lasts from 0.06–0.08 second (p. 46). However it causes prolongation of the sound in both the right and the left types of bundle branch block. The second sound, being shorter, is constantly split; the interval between the two phases of the split sound is from 0.03 to 0.04 (Fig 233).*

* If left ventricular hypertrophy and intraventricular block add their effects to that of left bundle branch block, the delay between the contractions of the two ventricles may reach 0.05 sec or more.[31] Then closing of the mitral (first valvular event of left heart) takes place at the time of opening of the pulmonic valve (second valvular event of right heart) because the normal isometric tension period (about 0.05 second) lasts as long as the pathologic interval between the contractions of the two ventricles. The phonocardiogram then reveals three groups of vibrations: the first is due to tricuspid closure, the second, to pulmonic opening plus mitral closure, the third, to aortic opening. A similar phenomenon with precession of left ventricular events occurs when right intraventricular block and right ventricular hypertrophy add their effects to that of right bundle branch block.

TABLE 27 ELECTROCARDIOGRAPHIC CHANGES IN RIGHT BUNDLE BRANCH BLOCK

Position of the heart	*Unipolar lead*	*Pattern of electrocardiogram*
Vertical heart (less common)	Rt arm	rSR inverted T
	Lt arm	rSR inverted T
	Lt leg	qRS upright T
	V1–V2	rsR inverted T
	V4–V6	qRS upward T
Horizontal heart	Rt arm	rsR inverted T
	Lt arm	qRS upward T
	Lt leg	rSr' or rSR upward T
	V1–V2	rsR or M inverted T
	V4–V6	qRS upward T

Carotid Tracing Jugular Tracing

The delay between beginning of first sound complex and rise of the carotid pulse (usually about 0 05 second) is increased and lasts from 0 07 to 0 08 second in *left bundle branch block* The incisura of the carotid pulse coincides

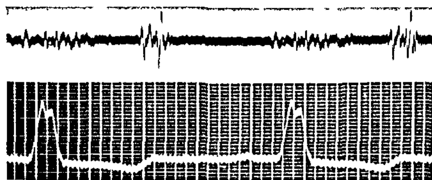


FIG 233 Phonocardiogram in a case of right bundle branch block Prolonged first sound split second sound

in normal subjects with the main vibration of the second sound In left bundle branch block it coincides with the second phase of the split second sound (Fig 234) [8, 18]

The rise of the carotid pulse has a normal relationship to the beginning of the first sound in *right bundle branch block* the incisura has a normal relationship to the first phase of the split second sound in this type of block

In *right bundle branch block* the study of the jugular tracing is of importance the peak of *v* is markedly delayed and occurs 0 08–0 14 second after the end of the second sound (or after the second phase of the second sound if this is split) In other words *v* which usually occurs 0 08–0 14

second after the second sound has the same relationship to the second phase of that sound when this is split (Fig 234)

Electrokymogram

This method may be applied to the study of pulmonic or aortic expansions In most cases, it may be applied not only to the study of left ventricular but also to that of right ventricular contraction Moreover, right atrial tracing is so deeply influenced by right ventricular contraction that the phases of right ventricular dynamics may be studied even by recording a right atrial border

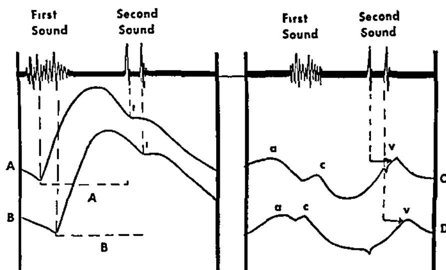


FIG 234 Scheme of the changes of the phonocardiogram carotid tracing and jugular tracing in bundle branch block with ventricular asynchronism At left delay of carotid pulse in left bundle branch block (B) At right delay of *v* wave of the jugular tracing in right bundle branch block (D)

tracing (p 192) The best technic is that of recording simultaneously a high left ventricular tracing and a pulmonic tracing, then, a right ventricular (or right atrial) tracing with an aortic tracing * The study may be completed by the simultaneous observation of the anterior (right ventricle) and posterior (left ventricle) surfaces of the heart It should be kept in mind that while direct comparison of the two ventricles has a decisive importance comparison of one ventricle with one pulse (or of the aortic and pulmonic pulses) is less

* If one has only a pick up unit the study should be made in the following way (1) aortic eky plus sound tracings (2) pulmonic eky plus sound tracings (3) left ventricular eky plus sound tracings (4) right ventricular (or right atrial) eky plus carotid and sound tracings (5) comparison of the four tracings using the sound tracing as timer It should be noted that use of the carotid tracing as timer is confusing because the carotid pulse is delayed in left bundle branch block

significant on account of possible interaction of peripheral factors which may accelerate or delay the valvular events

Normal time relationships between the two ventricles and the two large vessels were studied by the author and his co workers [9] and by Schwedel and co workers [8] In spite of slight individual and phasic variations, it can be assumed that the two ventricles and the two large arteries present pulsations which are either simultaneous or succeed each other within 0 01 second The most common is the precession of the right ventricle and of the pulmonic pulse by 0 003 second [9]

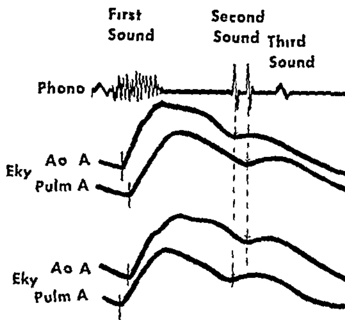


Fig 235 Scheme of the electrokymograms of the aortic and pulmonary arches in right (above) and left (below) bundle branch blocks with ventricular asynchronism

In about 30 per cent of the cases of *left bundle branch block*, left ventricular contraction and the aortic pulse were found delayed over the right ventricular and the pulmonic pulse by about 0 03 second (Fig 235) [31] If there is hypertrophy or intraventricular block of the left side the delay is greater Hypertension of the greater circulation increases the delay revealed by the rise of the pulse decreases that revealed by the incisura

In about 30 per cent of the cases of *right bundle branch block* the right ventricular contraction and the pulmonic pulse were found delayed over the left ventricular and the aortic pulse by about 0 03 second (Fig 235) [9, 31] If there is hypertrophy of the right ventricle or intraventricular block of the

right side, the delay is greater Hypertension of the lesser circulation increases the delay revealed by the rise of the pulse decreases that revealed by the incisura Systemic hypertension may partly neutralize the effect of right bundle branch block so that the delay is less apparent

In both types of block a prolongation of the phase of isometric relaxation was observed [9] A large percentage of cases of bundle branch block, whatever the side seem to present a bilateral delay of contraction[31] upon eky studies However, phonocardiographic studies reveal a much larger percentage of asynchronism

Conclusions

Certain data indicating asynchronism of ventricular excitation and contraction are common to both types, broadening and slurring of the QRS complex (ecg) prolongation of the first sound, and splitting of the second (phono)

Other data can be used for the diagnosis of side of the bundle branch block with the reserve that mechanical delay may or may not be present

1 *Left bundle branch block* The manifestations of left ventricular activity are delayed in comparison with those of the right ventricle delay of R in V5 V6 over R in V1 V2 (ecg), delay of the rise of the aortic over that of the pulmonic pulse (eky) coincidence of the carotid incisura with the second phase of the split second sound (phono, carotid tracing)

2 *Right bundle branch block* The manifestations of right ventricular activity are delayed in comparison with those of the left ventricle existence of S1 delay of R in V1 V2 over R in V5 V6 (ecg) delay of the rise of the pulmonic over that of the aortic pulse (eky), delay of the *v* wave of the jugular tracing over the second phase of the split second sound (phono, jugular tracing)

INTRAVENTRICULAR BLOCK

Electrocardiogram

In diffuse intraventricular block QRS is severely prolonged and may last more than 0 15 second Bundle branch block or ventricular hypertrophy may be contributory factors

In left intraventricular block a small Q may be observed in the chest leads V5 V6 This is particularly significant because the delay of conduction in the left branch of the bundle should cause disappearance of a septal Q Septal infarction should be excluded before explaining this Q as caused by intraventricular block

It has been suggested that certain changes of the electrocardiogram indicate block of the stimulus in some definite layers of the ventricular wall *Notchings* of the ascending branch of QRS have been interpreted as due to block in the

subendocardial layers while notchings of the descending branch of QRS have been explained as due to block in the subepicardial layers * *Focal* block is revealed by a broadening of QRS only in one or two chest leads and not in the others This is explained as due to local conduction disturbance

WOLFF PARKINSON WHITE SYNDROME

This syndrome[43] should be kept apart from the more common types of intraventricular block because it is due to preexcitation of the ventricles and not to delay of ventricular excitation It occurs mostly in young males who may present attacks of paroxysmal tachycardia but are otherwise normal However cases where this syndrome is associated with rheumatic congenital or coronary heart disease have been described

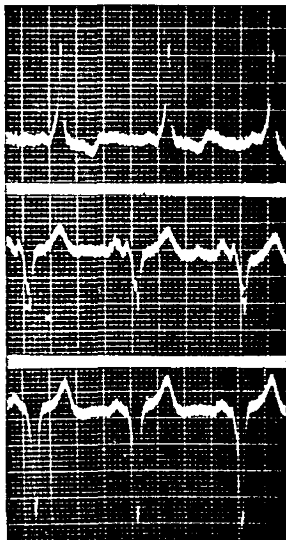


Fig 236 Ecg of a case of Wolff Parkinson White syndrome

Electrocardiogram

The electrocardiogram presents a short P R interval (0 08–0 10 second) while QRS is increased in duration (0 10–0 12 second) and is slurred It is typical that the first limb of QRS is slurred and slow while the second limb is rapid and normal (Fig 236) The precordial leads usually fail to show those changes which are common in bundle branch block

* Seghers[33] denies the existence of bundle branch block He admits (1) a septal block causing the pattern of ventricular preponderance (2) a peripheral block in the ventricular wall He further admits that the peripheral block should be subdivided into (1) subepicardial (descending branch of QRS is notched) (2) intermediate or parietal (ascending branch of QRS is notched) subendocardial (shift of the electric axis of R toward the normal side early intrinsicoid deflection in the chest leads on the side of the block)

During the attacks of tachycardia (atrial tachycardia), the ecg often presents normal PR and QRS intervals However, attacks of ventricular tachycardia or atrial fibrillation may also occur

Phonocardiogram

In general the phonocardiogram fails to show prolongation of the first sound, like in bundle branch block A case observed by the author presents a crescendo type of first sound which had been erroneously interpreted as a presystolic murmur Only rare cases present a split second sound

Electrokymogram

The most common finding is that of delayed contraction of both ventricles[30] as revealed by simultaneous aortic and pulmonic tracings with an electro cardiogram Less common finding is that of ventricular asynchronism[30]

MYOCARDITIS

Acute and subacute myocarditis may be viral bacterial or parasitic or may follow an allergic reaction Vitamin deficiency states and severe neuropathies and myopathies are also frequently accompanied by degeneration and failure of the myocardium

Electrocardiogram

The findings vary according to the more or less widespread type of lesion They consist of

1 Changes of rate
2 Disturbances of the rhythm due to increased excitability (premature con tractions atrial flutter or fibrillation atrial or ventricular tachycardia)
3 Disturbances of the rhythm due to altered spreading of the stimulus (pro longed P R, s a or a v block b b block i v block)
4 Abnormality of repolarization (changes of S T and T) In certain cases the electrical pattern may simulate myocardial infarct[11]

Phonocardiogram

The most common findings are

1 Apical systolic murmur
2 Triple or quadruple rhythm
3 Prolonged faint and low pitched first sound
4 Split second pulmonic sound

Arterial Tracing

There may be pulsus alternans or irregularities of the pulse caused by cardiac arrhythmias

Electrokymogram

Certain cases having a severe but circumscribed lesion of the left ventricle may present the pattern of dynamic aneurysm (p 452) over the border of the left ventricle This was found in 1 case of the author[10] and in 1 case of Clagett [9] both confirmed by autopsy

Patients with myasthenia gravis may present this pattern [9] which may disappear after treatment It is likely that more cases of myocarditis myo cardial degeneration or myocardial fibrosis presenting this abnormality will be described in the future

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CHAPTER 49

Coronary Heart Disease

(INCLUDING MYOCARDIAL INFARCT)

The term coronary heart disease is applied to lesions of the arterial system of the heart and to the resulting changes of the myocardium It is also applied to the clinical pictures caused by these lesions

Polyarteritis nodosa a process attributed to an allergic mechanism frequently involves the coronary arteries Thromboangiitis obliterans (Buerger's disease) may cause coronary stenosis and occlusion in young people However arteriosclerosis is the most common cause of coronary lesions and is present in over 90 per cent of cases

Coronary insufficiency is a condition in which the need of the myocardium for blood and oxygen is greater than the amount which can be supplied Coronary insufficiency does not occur when both blood supply and blood demand are reduced as in the senile heart and in certain forms of vascular collapse The most common manifestation of coronary insufficiency is cardiac pain, the so called angina pectoris

Coronary heart disease may lead to various clinico anatomic pictures (1) myocardial infarction (2) cardiac pain (angina pectoris) (3) senile heart (4) congestive failure

Coronary disease is frequently accompanied by decreased blood supply to the myocardium (ischemia) The latter may be followed by damage (injury) to the heart muscle and if severe by its necrosis (death) Consequently three basic electrocardiographic patterns have been recognized ischemia injury, and death (Fig 237)

1 *Pattern of ischemia* According to widely accepted studies, the spreading of the stimulus within the ventricular wall takes place from the endocardial toward the epicardial layers The front of the wave of depolarization is preceded by positive and followed by negative ions Therefore, an epicardial electrode records the wave of depolarization as a positive deflection while an electrode introduced into a ventricular cavity records it as a negative deflection Repolarization of the ventricular wall in the human heart has features which are different from those of other species On account of the slow repolarization of the subendocardial layers the front of repolarization starts from the subepicardial layer and moves from there toward the endocardium As the front of repolarization is preceded by negative ions and is followed by positive ions an epicardial electrode records a positive T while a cavity electrode records a negative T Ischemia affects chiefly the subepicardial layers and slows down their process of repolarization Thus, the vector of repolarization of the ischemic ventricle proceeds from the cavity toward the surface and a negative T is recorded by an epicardial electrode

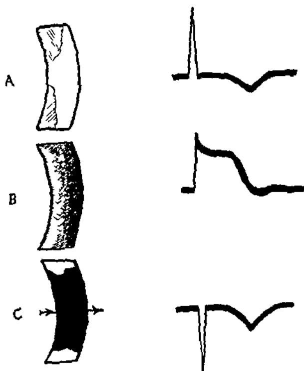


FIG 237 Electrocardiographic patterns in coronary heart disease *A* ischemia *B* injury *C* necrosis

2 *Pattern of injury* Understanding of this requires resort to a concept which has been used several times in the past and which is still useful that of

the monophasic waves According to it the electrocardiogram is the result of an algebraic sum of two monophasic waves One due to the electrical processes of the subendocardial layers would be positive the other, due to the electrical processes of the subepicardial layers would be negative The QRS T is due to the precedence of depolarization in the subendocardial layers to the precedence of repolarization in the subepicardial layers Injury to the subepicardial layers would decrease the potentials of the latter and cause a predominance of the positive displacement which is especially noticeable in the S T interval On the contrary injury to the subendocardial layers would decrease the potentials of the latter and cause a negative displacement of S T If the injury is caused to the layers of the posterior wall of the heart an anterior electrode would record a displacement but in the opposite direction (depression in posterior subepicardial injury rise in posterior subendocardial injury) Both the chest and the limb unipolar leads employed in clinical studies use an exploring electrode which is at some distance from the epicardium, therefore, a mixed pattern of injury plus ischemia (raised S T and inverted T wave) is recorded, unless the area of injury is large

3 *Pattern of necrosis* The destruction of an area of ventricular muscle results in the disappearance of the electrical forces due to the activity of that area during the process of excitation As a result the dead tissue acts like 'an electrical window and the electrode records the negative potentials of the ventricular cavity Necrosis or death of the tissue is revealed by a deep QS complex followed by an inverted T wave if an electrode is placed over the epicardium * Unipolar limb leads (p 231) or unipolar chest leads (p 232) may register a mixed pattern of necrosis injury, and ischemia, if the area of necrosis is small Then, a deep Q wave an elevated S T, and a small inverted T wave are observed On the other hand if the area of necrosis is large, the unipolar leads record typical cavity potentials e g a pure pattern of necrosis Whenever the necrosis does not involve the entire thickness of the ventricular wall potentials from both the damaged and the healthy tissue reach the electrode Then, it is impossible to distinguish between prolonged diffuse ischemia plus some degree of injury, and partial necrosis except by the duration and evolution of the pattern

MYOCARDIAL INFARCTION

Myocardial infarction may be the result of acute coronary insufficiency, coronary occlusion or coronary embolism The most common occurrence is that of the gradual occlusion of an arteriosclerotic vessel by the concentric

* Put in a different way the normal vector of electrical forces within the ventricular wall is directed from the endocardium toward the epicardium whenever necrosis of the wall occurs an epicardial electrode records currents directed away from it which are transcribed as negative waves

proliferation of the intima Another possibility is that of a sudden occlusion because of intramural hemorrhage or thrombosis of a coronary vessel

Acute ischemia is followed by severe functional changes in the myo cardium The ischemic area becomes cyanotic and its contractions weaken rapidly Then the area cannot oppose the sudden rise of ventricular pressure which takes place during each contraction and dilates in systole forming a ventricular aneurysm There is a sudden fall of pressure in both the aorta and the pulmonary artery which may be partly compensated within a few minutes The area surrounding the infarction becomes hyperexcitable Stimuli arising in this area may cause premature beats ventricular tachycardia atrial flutter or fibrillation, or ventricular flutter The sudden ischemia may cause a v block or bundle branch block

Electrocardiogram

The typical changes revealed by the electrocardiogram are those of *ischemia injury and necrosis as outlined above (Fig 237) Different pictures* occur according to the localization of the infarct The older division of infarcts into anterior (Q1 T1) and posterior (QT T3) may be maintained for large in farcts Otherwise it should be replaced by a more precise description of localization such as anteroseptal anterolateral posterolateral or posterobasal A high anterolateral or posterolateral infarct may also occur [21]

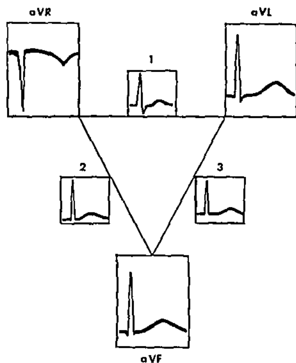
The typical evolutionary changes of the tracing are well known They consist of the appearance of a QS type of initial complex (or a deep Q fol lowed by R) and an inverted T wave (Fig 242) In the process of healing, the negative T wave becomes deeper at first, then decreases and may become isoelectric and even return to norm These changes are due to the spreading toward definite areas of the body of negative cavity potentials recorded through the necrotic tissue It could be said that those areas look inside the ventricle through an electrical window

The *anteroseptal variety* does not affect the limb leads even though it causes important variations in V1, V2 and V3 and occasionally in Ve (Fig 238)

The *anterolateral variety* affects typically the unipolar lead of the left arm (aVL) This causes the well known changes in the standard lead 1 Reciprocal changes take place in the right arm lead The chest leads V4 to V6 reveal the infarction and may permit a gross evaluation of its size (Fig 239)

Both in the *posterolateral* and *posterobasal varieties* the typical inversion of the initial complex and of the T wave again due to the "electrical window is recorded best by the unipolar lead of the left leg (aVF) A reciprocal change is recorded by the left arm The resulting balance is revealed chiefly by the standard lead 3

In the *posterolateral variety* the posterior chest leads V7 to V9 and the



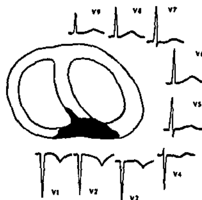
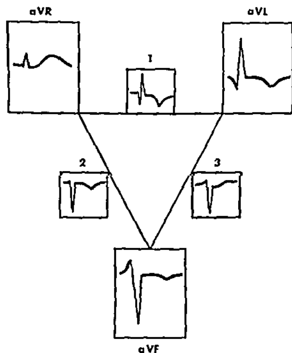


FIG 238 Ecg pattern of old anteroseptal infarct



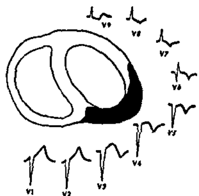


FIG 239 Ecg pattern of old anterolateral infarct.

esophageal lead (VEs) reveal typical changes (Fig 240) In the *posterobasal variety* the unipolar lead of the xiphoid process (Ve) reveals the typical picture (Fig 241) On the other hand the chest leads V1 to V4 present reciprocal changes in both varieties

Infarcts may occur in any part of the myocardium including the atria and the septum They may occur near the epicardium or near the endocardium they may be transmural in which case they are like a combination of the previous two types, they may be intramural because small In the last instance the

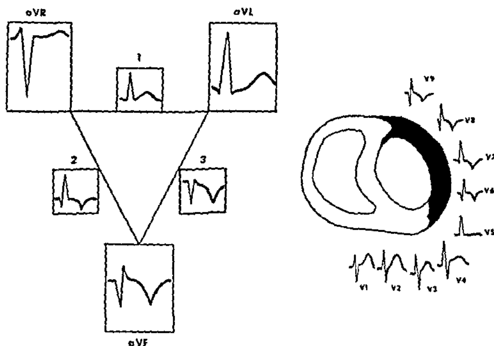


Fig 240 Ecg pattern of old posterolateral infarct

electrical forces around the infarcted area are equal and opposite so that no pattern of injury or necrosis may be recorded if on the other hand a large area of ischemia surrounds one of these small infarcts a pattern of ischemia is recorded

The direction of the S T displacement may give data on the location of a myocardial lesion because the changes of the S T segment are different according to the depth and location of the lesion [1] Elevation of S T is found in anterior chest leads when there is an *anterior subepicardial injury* A *posterior subendocardial injury* or an *injury of the septum* on the left ventricular side is also revealed by elevation of S T in these leads

Electrocardiographic Diagnosis

The unipolar limb and chest leads are of great value for the diagnosis [14, 15]

An *upward T wave in aVR* can be found in the following conditions (1) *left ventricular hypertrophy, (2) left bundle branch block, (3) myocardial infarction* (4) after digitalis

An *inverted T wave in aVL* can be found in the following conditions (1) normal heart in vertical position, (2) left ventricular hypertrophy or left

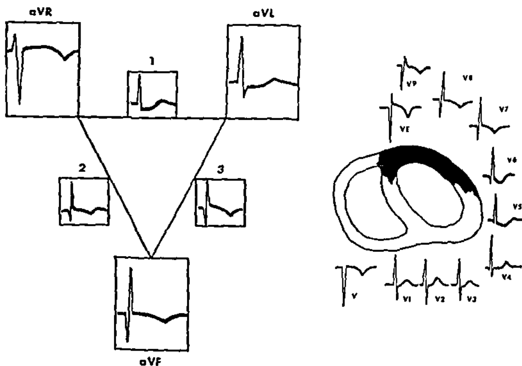


FIG 241 Ecg pattern of old posterobasal infarct

bundle branch block in a horizontal heart, (3) anterior myocardial infarct (4) after digitalis

An *inverted T wave in aVF* can be found in the following conditions (1) normal heart in horizontal position (2) right ventricular hypertrophy (cases with inverted T2 T3) (3) right bundle branch block with a horizontal heart (4) left bundle branch block with a vertical heart (rare) (5) posterior myo *cardial infarct, (6) after digitalis*

Inversion of the T wave in the chest leads together with changes of the QRS complex permits localization of the infarction (Table 28)

ATRIAL INFARCT This rare lesion is revealed by abnormalities of the P wave premature beats atrial fibrillation wandering pacemaker or nodal rhythm

TABLE 28 INVERTED T IN THE CHEST LEADS

At the right of the transitional zone	Normal heart (especially infants and children) Right ventricular hypertrophy Right bundle branch block Anteroseptal infarct*
At the left of the transitional zone	*After digitalis* Left ventricular hypertrophy Left bundle branch block Anterior infarct Infarcts extending to the lateral aspect of the heart (anterolateral plus posterolateral)

* This may extend to the first portion at the left of the transitional zone

Later, either elevation or depression of the P Q interval with a cove shaped aspect has been observed There is elevation of P Q in Lead 1 when the *left* atrium is involved in Lead 3 when the *right* atrium is damaged [21]

Later in the stage of inversion of the T atrial wave there may be some slurring of QRS because repolarization of the atria (Ta) occurs during the QRS complex

ATYPICAL FORMS MULTIPLE INFARCTS Atypical electrocardiographic patterns are found in about one third of the cases with infarct Some are due to pre existing intraventricular block or older infarcts some to gradual production of necrosis with successive involvement of anterolateral and posterolateral parts of the left ventricle In such instances the first stage may not be apparent

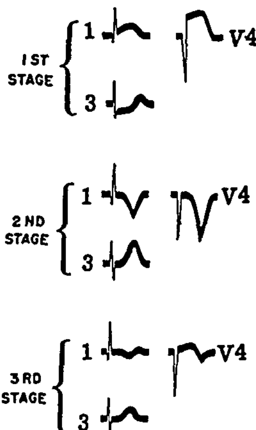


FIG 242 Electrocardiographic changes in the various stages of evolution of an anterior infarct

Multiple infarcts are common and may consist of various combinations of recent and old infarcts In some cases an inverted T wave in all leads has been observed (type Tn) [6] This may be due to (1) massive infarct involving both the anterior and posterior walls of the left ventricle (2) anterior infarct in patients already having an inverted T3, (3) posterior infarct in patients already having an inverted T1

Serial electrocardiograms in successive infarcts may reveal either the Tn type of evolution or changes of T this wave first assumes a frank anterior, then a frank posterior type in spite of previous abnormalities

Phonocardiogram

The most typical change is the disappearance of the rapid vibrations of the first sound This presents a prolonged series of slow vibrations which are frequently small but may be of normal height After the acute stage it is possible to observe large diastolic sounds These additional sounds are heard only if they are very loud on account of their low pitch (Fig 243 B)

Low Frequency Tracings (Cardiograms)

Cardiographic tracings are obtained with difficulty in the acute stage of myocardial infarction because of the weak cardiac action When a good tracing is obtained a slow development of all waves is frequently observed (Fig 243 A) This is due to the existence of an area of soft tissue in the left ventricle By absorbing part of the energy of contraction, this area prevents a rapid rise of pressure and a rapid closure of the mitral valve Therefore the first sound becomes altered and the low frequency vibrations of the chest also become slow (Fig 243)

Roentgenkymogram

The abnormal movements of the left ventricular wall following myocardial infarction have been repeatedly investigated by means of roentgenkymography

The most common abnormalities described were diminution or absence of pulsation in a segment of the left ventricular contour, systolic expansion over the lower left contour, and diastolic splintering [10, 11, 24, 25]

Electrokymogram

Electrokymography permits an accurate study of these movements and gives tracings which may have diagnostic value [7, 13, 16, 17, 22]

Several abnormal patterns can be observed in either systole or diastole (Fig 244) [16] However some of the abnormalities can be found in other conditions or can be simulated by improper application of the slit Therefore they have no diagnostic significance as to myocardial infarct Some of the diastolic abnormalities are connected with an abnormality of systole and represent a necessary consequence of the latter (return of the wall after abnormal pulsation) Furthermore some of these deviations from the normal tracing, and particularly those extending over a large portion of the left ventricular contour may be favored by positional changes of the heart

Two definite abnormalities of the systolic wave should be considered as

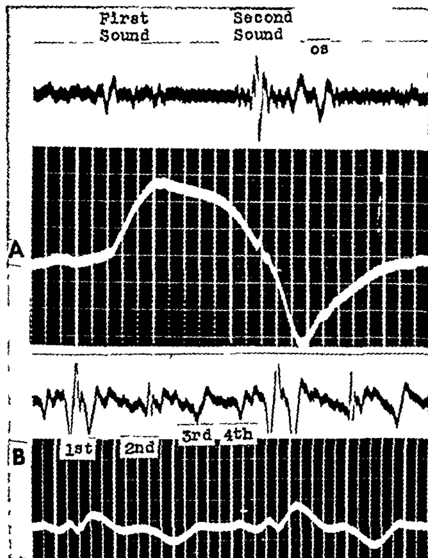


FIG 243 Two cases with coronary heart disease and old infarcts (*A* anterior, *B* posterior) Both the stethoscopic phonocardiogram and the *low frequency tracing at the apex reveal a slow development of all* vibrations

indicative of localized myocardial damage in the majority of instances this being identical with myocardial infarct

1 *Reduced amplitude of the ventricular wave or disappearance of this wave in a circumscribed region of the left ventricle* Whenever the surrounding areas present large waves this sign is definitely related to infarction As that area is functionally (and usually also anatomically) excluded from participat

ing in active contraction the author suggested the name of *local paralysis* for the phenomenon thus revealed by electrokymography[16]

2 *Inverted pulsation (paradoxical pulsation) of a circumscribed area of the ventricular myocardium* In typical cases this inverted pulsation assumes the aspect of a *plateau*, indicating that the inert wall is passively distended by intraventricular pressure (Figs 244, 245 and 246) This type of pulsation

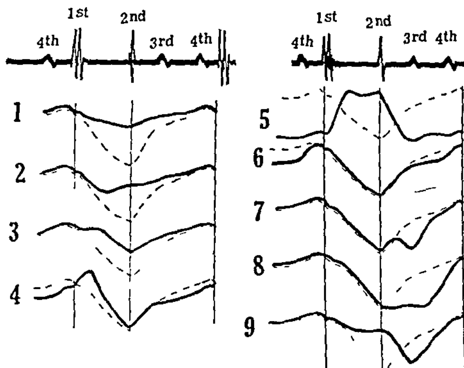


Fig 244 Abnormalities of the ventricular electrokymogram (continuous line) compared with the normal tracing (dotted line) *1* Decreased amplitude of contraction *2*, early end of contraction *3* late onset of contraction, *4*, early systolic distention followed by normal contraction *5* *systolic distention (dynamic aneurysm)* *6* marked presystolic distention *7*, deep early-diastolic rebound *8* late diastolic distention *9*, decreased amplitude of contraction and deep early-diastolic drop (From Luisada and Fleischner, *Acta Card*)

may be associated with the existence of a well defined bulge of the ventricular profile on chest films In such cases the name of *ventricular aneurysm* is commonly used In other cases the bulging occurs only in systole while no bulge is present in diastole This phenomenon occasionally observed on fluoroscopy is hardly noticeable on roentgenograms, these even if taken at the maximum of systole would show but a minute projection hardly detectable without simultaneous observation of the two opposite motions It

should be kept in mind that the dynamic significance of such an inverted pulsation is similar to that of an aneurysm The wall distends in systole absorbing part of the dynamic effort of the normal myocardium it collapses in diastole disturbing the normal filling and spilling its retained blood into the rest of the ventricular cavity This similarity may justify the term *dynamic aneurysm* suggested for the inverted type of pulsation without a permanent bulge of the left ventricle[10]

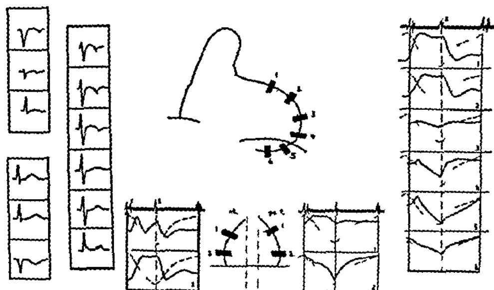


FIG 245 Locations of the slit and reconstruction of the structural changes in a case with multiple myocardial infarction Large dynamic aneurysm extending from the upper part of the lateral wall to the lower part of the anterior wall Local paralysis in the middle lateral and posterior walls (From Luisada and Fleischner *Acta Card*)

The myocardial damage may not be revealed by the electrokymogram in small infarctions on the other hand it may be more extensive than forecast by the electrocardiogram in cases with multiple old infarcts

Landowne[13] has remarked that the pattern of local paralysis indicates that the damaged area fails to move inward like the rest of the ventricular wall Thus it is equivalent to a moderate relative expansion If this explanation is accepted local paralysis would be equivalent to an initial ' dynamic aneurysm

ANGINA PECTORIS

Increasing knowledge in the field of coronary heart disease has changed our understanding and definition of angina pectoris Fifty years ago this name

was applied to any case of coronary heart disease with cardiac pain cases of myocardial infarct are now excluded Therefore angina pectoris is simply cardiac pain due to coronary insufficiency not followed by an infarct and the expression cardiac pain might be substituted for angina pectoris

Even if hemodynamic chemical or vasomotor elements are involved there is no doubt that a structural lesion of the coronary system is present in the great majority of cases This lesion may be severe and extensive in some patients In others it is so limited that it acts only as a trigger mechanism' favoring reflex coronary ischemia

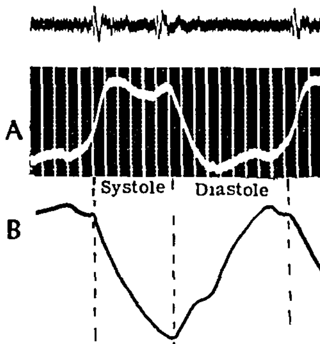


FIG 246 A case of old anterolateral infarct (electrocardiogram) with anterolateral dynamic aneurysm (electrokymogram) Phonocardiogram *ventricular eky (A) and for comparison a normal* ventricular eky (B) (From Luisada *Medical Clinics of North America* Courtesy of the Saunders Co)

Electrocardiogram

In patients with frequent attacks of cardiac pain the electrocardiogram should be taken between the attacks and also during an attack In doubtful cases the study should be repeated after controlled exertion (Master s two step exercise test[10] [0] (p 252) inhalation of oxygen poor gas (according to Levy s technic[11]) (p 252) or inhalation of amyl nitrate[6] p 254) This leads to division of the patients in four groups [1]

1 The electrocardiogram between attacks reveals severe abnormalities indicating coronary and myocardial lesions (no functional tests should be performed) A predominant structural component is at the basis of the attacks

2 The electrocardiogram between attacks reveals only slight abnormalities However functional tests cause typical changes of the electrocardiogram The structural lesion is mild and functional components contribute to the attack

3 The electrocardiogram is normal between attacks Functional tests cause

typical changes Even though a predominantly functional mechanism is involved, a slight structural lesion is to be suspected

4 The electrocardiogram is normal both between attacks and following functional tests The pain is probably not related to the heart

CORONARY HEART DISEASE WITHOUT CARDIAC PAIN

Coronary heart disease can be followed by severe changes in the myocardium without causing cardiac pain This is probably due to uniform and widespread fibrosis of the myocardium with moderate atherosclerosis of the

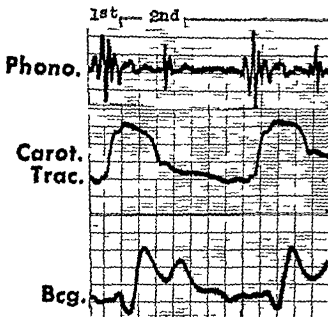


FIG 247 Abnormal ballistocardiogram (Bcg.) in a case of coronary heart disease

larger coronary arteries If the fibrosis of the myocardium precedes or accompanies the hardening of the larger vessels cardiac work and blood demand are decreased without disproportion to the amount of blood supplied by the larger coronary vessels As a result of myocardial fibrosis cardiac reserve is decreased Whenever the lesion is moderate the picture of *senile heart* is observed On the other hand if severe lesions occur within a short time or damage is extensive *heart failure* may follow

Ballistocardiogram

The ballistocardiogram of patients with coronary heart disease is frequently abnormal (Fig 247) [1, 9, 18] The possible abnormalities are

1 H wave of amplitude equal or higher than that of the J wave (early M type)
2 Absence of the J wave
3 J wave M shaped at its peak (late M type)
4 J wave late in systole and deep K wave (late downstroke type)
5 R J interval longer than 0 29 second
6 L wave and afterwaves accentuated
7 Upward bowing of the J K segment

It is still open to question which part is played by hypertension or ather osclerosis of the aorta and large vessels in causing these changes As they are similar to those observed in elderly individuals (pp 292 and 487),[9] they have a definite meaning only if observed in younger subjects It is also open to question whether the abnormalities of left ventricular contraction causing these changes might not be more clearly detected by the study of the electrokymogram of the left ventricle and the aortic arch or simply by recording a carotid tracing

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CHAPTER 50

Diseases of the Pericardium

ACUTE AND SUBACUTE PERICARDITIS

Two different stages or phases of pericarditis can be recognized *dry pericarditis* and *pericarditis with effusion* Either may result in the formation of adhesions

Electrocardiogram

The ecg may be completely normal in the phase of dry pericarditis, except for sinus tachycardia However electrocardiographic changes consisting of low voltage of the QRS complex and changes of S T and of the T wave, may be observed (Fig 248)

Three different electrocardiographic stages are recognized [7, 8] acute sub acute, and chronic

1 *Acute stage* The pattern is characterized by elevation of S T in both the limb and the chest leads This elevation is maximal if the exploring electrode is placed over the area where a friction rub is audible

2 *Subacute stage* The S-T elevation gradually decreases and a moderate inversion of T in the limb and the chest leads becomes apparent The T waves are not cove shaped and the contour of the ascending limb of the elevated S-T segment may be concave

3 *Chronic stage* The inverted T wave may last for a long time and even

become permanent However it usually becomes flat and then returns to normal within a few weeks

In general the maximum inversion of T occurs between the sixteenth and the forty third day of disease, it may involve only one or two leads with merely a depression in the others No deep Q wave is ever observed during the changes of S T

Phonocardiogram

The *friction rubs* are represented on the sound tracing by vibrations of various pitches with those of higher pitch prevailing These vibrations are grouped in either two or three phases of the cardiac cycle The most common are those which occur during presystole and systole (to and fro rubs) the latter being louder a third group may take place in early diastole (Fig 249) [8] The systolic rub is due to friction of the pericardium during ventricular contraction, the presystolic rub is caused by friction of the pericardium during atrial contraction the early diastolic rub is caused by friction of the pericardium during rapid ventricular filling and is the least loud on account of the slower motion causing it

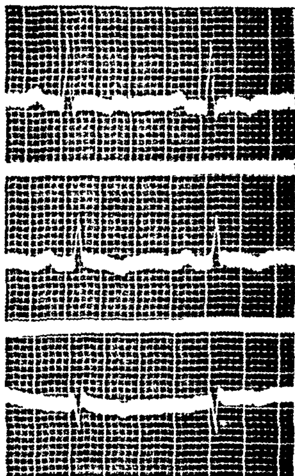


FIG 248 Electrocardiogram of pericarditis

Increased compression with the microphone increases the loudness of the vibrations probably through modification of the acoustic properties of the microphone The vibrations of the rubs are more easily recognized when no endocardial murmurs are present

Electrokymogram

The electrokymogram of both left and right cardiac borders reveals extremely reduced pulsations in pericarditis with effusion In certain cases no

pulsations can be recorded Theoretically the pulsations of the aortic arch should be normal This contrast should indicate that reduction of the ventricular waves is due solely to masking because of the presence of fluid This fact previously described by means of roentgenkymography, is actually observed in certain cases in others however cardiac output is reduced by the compression and by myocardial damage and the aortic pulsations are small After tapping of the fluid and introduction of air an air fluid level is formed in the pericardial sac Placing the slit of the phototube across the fluid reveals pulsations of a ventricular type on the left of an atrial type on the right

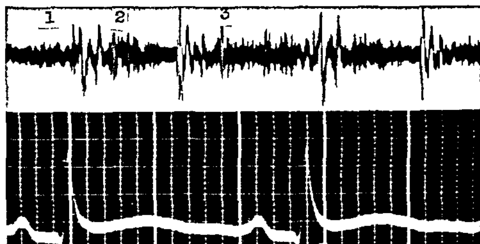


FIG 249 Phonocardiogram of pericarditis Three separate groups of high pitched vibrations in presystole systole and early diastole (*1 2 3*), indicate the friction rubs

Cardiomanometry Venous Pressure

The right atrial tracing may reveal increase of pressure but no abnormal pattern The right ventricular tracing may reveal a certain increase in the magnitude of the diastolic pulsations Venous pressure tracings may show a high level and a decreased amplitude of the various waves

Jugular Tracings

It is typical to obtain tracings with poor amplitude of all waves This is in contrast with the engorgement of the jugular veins

CHRONIC CONSTRICTIVE PERICARDITIS

The frequent involvement of the pericardium in lesions of the mediastinum pleurae and diaphragm and the connection between the clinical picture and

the extension and location of the adhesions explain the common name of mediastino-pericarditis Compression of various structures possible involvement of the endocardium and pleural or peritoneal lesions may determine different clinical pictures

Electrocardiogram

Disturbances of cardiac rate and rhythm are sometimes present because of myocardial or coronary lesions or because the traction of adhesions favors the occurrence of premature beats paroxysmal tachycardia or atrial flutter Atrial fibrillation may occur However the most usual findings are regular sinus rhythm low voltage in the limb leads low flat or inverted T waves in one or more limb leads occasional splintering of the ventricular complex Right axis deviation has been observed when there is predominant compression of the left ventricle [2] S T may be depressed Changes of the P wave consisting of broadening notching and increased voltage have been noted [19] these have a certain value whenever QRS has a low voltage in all leads

The study of the deviation of the electric axis in different positions may reveal that the heart is fixed and is not displaced by gravity a fact which may be used in the differential diagnosis with congestive failure [5] This sign is observed when extensive internal and external adhesions are present (Fig 250) and not in cases having only internal adhesions

Deep respiration may cause premature beats a v block or s a block

Phonocardiogram

It is possible to note increased loudness of the second pulmonic sound This is evidence of constriction of the left cardiac chambers followed by higher pressure in the pulmonary circulation [2]

A triple rhythm of either the atrial or ventricular type is frequently recorded A high pitched diastolic click is recorded frequently in calcification of the pericardium and has a wide diffusion This occurs at the time of rapid filling and is the pathologic equivalent of a third sound

A typical finding is that of a systolic click or snap This is a high pitched vibration *at or after the middle of systole frequently followed by a few* smaller vibrations (Fig 251) Cases where two or three small extra sounds are present in systole may occur An abnormal depression or an additional wave of the cardiogram frequently coincides with the click

Simultaneous tracings of the jugular veins and of the radial pulse reveal the frequent existence of Kussmaul s phenomenon inspiration causes swelling of the jugular veins and simultaneous weakening (or even disappearance) of the radial pulse (pulsus paradoxus) (Fig 252) Disappearance of the pulse during expiration a more unusual phenomenon is called *Riegel's sign*

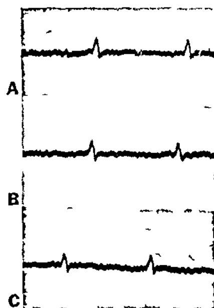


Fig 250 Absence of changes of the electric axis in a subject with adhesive pericarditis Lead 1—(A) supine (B) on the right side (C) on the left side



Fig 251 Systolic click (*SC*) in a patient with pleuro pericardial adhesions

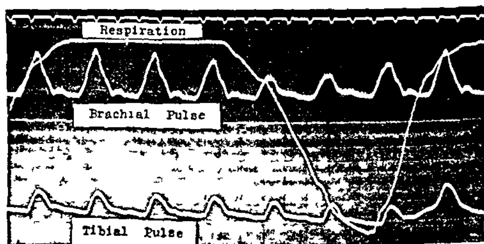


Fig 252 Kussmaul phenomenon Inspiratory decrease of the arterial pulse

Pneumocardiogram

An abnormal phenomenon consisting of the inversion of the normal systolic waves has been described in some patients [11]

In the authors cases[14] *the waves were of normal type but smaller than in* normal subjects The difference in the findings may be explained by different *criteria used in the selection of the cases*

Electrokymogram

A typical diagnostic sign of constrictive pericarditis has been described by Gillick[8] and confirmed by McKusick [16] the border tracing of the left ventricle presents a sharp and deep drop followed by a rapid rise There is a flat line during most of diastole and no evidence of the normal rebound which

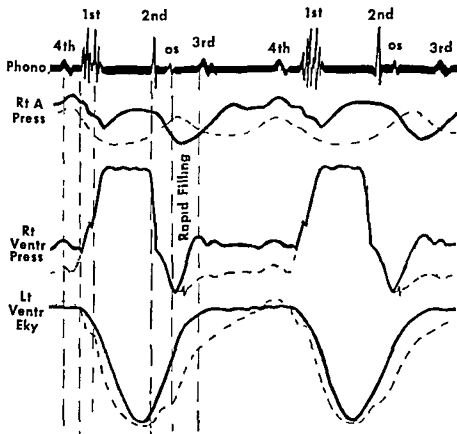


FIG 253 Scheme of the V type of electrokymogram and of the typical pattern of intracardiac tracing in constrictive pericarditis

marks the end of isometric relaxation period Also the secondary waves due to positional and rotational movements are absent This pattern has been called the *V type of ventricular contraction* Ventricular filling is very rapid and comes to an abrupt end (Fig 253)

Less constant features are

1 A late systolic plateau in the ventricular border tracing (impediment to systolic emptying)

2 A small rise above the base line of the diastolic level at the end of rapid filling
3 Long flat, diastolic intervals between the pulse waves of the pulmonary artery (constriction of the pulmonary artery)
4 Damping or obliteration of the pulsations of the right atrial border
5 Prominent pulsations of the superior cava, occasionally with a diastolic plateau

Cases studied by the author presented, on the other hand only a severe decrease in amplitude of pulsations over all structures It is likely that the difference results from the observation of different types of patients those studied by Gillick were cases selected for surgery while those studied by the author were unselected cases

Cardiomanometry

Pressure measurements have diagnostic importance in certain cases[3, 9, 23] Mean right atrial pressure is elevated and diastolic atrial pressure is also high The pulsations of the right atrium have a pattern resembling an M due to equal height of the two highest peaks This occurs even in atrial fibrillation

The systolic pressure of the right ventricle is usually within normal limits, however ventricular diastolic pressure is far above the zero line (Fig 253) There is a steep rise in early diastole, the tracing reaches its highest diastolic level at about one half of diastole, then forms a diastolic plateau Pericardial clicks were recorded at the end of isometric relaxation Coarser, inaudible vibrations were often noted at the beginning of diastole All the above findings were never observed in pericarditis with effusion polyserositis or congestive failure[9]

Abnormally high pressure in the superior cava with normal or low pressure in the right atrium is evidence of pericaval or periatrial constriction

High pressure in the right ventricle and pulmonary artery has been interpreted as evidence of constriction of the left ventricle and atrium[2] However both mitral stenosis and left heart failure should be excluded before attributing a decisive value to these data

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CHAPTER 51

Diseases of the Aorta and Pulmonary Artery

COARCTATION OF THE AORTA

There are two main types of coarctation

1 A narrowing of the aortic isthmus slightly above the opening of the ductus arteriosus This is a truly congenital lesion and is present since birth (so called "infantile type or preductal type of coarctation) It may be associated with patency of the ductus and may reach the extreme grade of aortic atresia

2 A narrowing of the aorta at or slightly below the level of the ductus arteriosus The lesion is congenital but may become gradually more severe during life (so-called 'adult type or postductal type of coarctation)

Electrocardiogram

This tracing may reveal the existence of left axis deviation There may be evidence of left ventricular hypertrophy left intraventricular block or both On the other hand coarctation plus patency of the ductus is usually followed by right ventricular hypertrophy and strain

Phonocardiogram

A systolic murmur caused by the narrowing of the aorta is frequently recorded The vibrations of this murmur have usually a diamond shaped aspect but may be nondescript in type This murmur is recorded best over

the base especially at the left of the sternum It may be recorded even better at the back, between left scapula and spine, as proven by Wells *et al*[9] When the murmur is louder at the back an important differential datum with any valvular defect is obtained An early diastolic murmur similar to that of aortic insufficiency is recorded in most cases at the back even though it may not be heard with a stethoscope[9] It is caused by the blood flowing through the narrow section of aorta during diastole

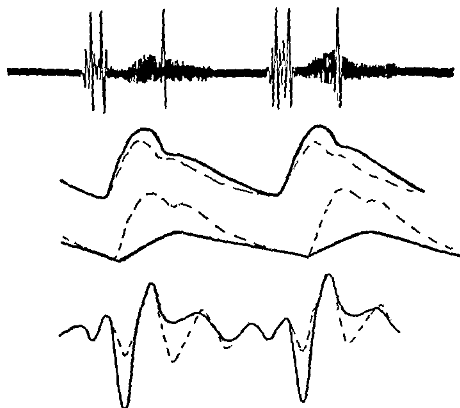


Fig 254 Scheme of changes in coarctation of the aorta Phonocardiogram over the back, *brachial and femoral tracings ballistocardiogram*

Pulse Tracings

Pulse tracings reveal that the femoral pulse which normally is simultaneous with the brachial pulse occurs some time later[1, 9] In general the rise of the femoral pulse in these cases is only slightly delayed over that of the brachial but the femoral peak occurs from 0 15 to 0 20 second later than the brachial peak The femoral pulse has a small amplitude and a slow ascending branch (Fig 254) It may be so small that no record is possible

Pressure Tracings

Direct or indirect pressure tracings of the brachial artery reveal the exist ence of normal or elevated arterial pressure in the upper extremities * Indirect pressure tracings of both lower extremities may not be possible on account of too small amplitude of the pulse If obtained they reveal a low pulse pressure Direct pressure tracings are possible even in cases where no pulsation is felt Then a pressure level of 60–90 mm of mercury may be obtained with small oscillations or none at all This is evidence of a nearly continuous blood flow, partly through the narrow section and partly through anastomotic arterial vessels

Roentgenkymogram Electrokymogram

Roentgenkymography shows a sharp contrast between the pulsations of the aortic arch and those of the descending aorta

Electrokymography also shows that the densogram of the aortic arch has large pulsations while that of the descending aorta has small pulsations or none at all (Fig 254) [8]

Ballistocardiogram

The ballistocardiogram presents typically the absence of the K wave (Fig 254) [7] This pattern which has diagnostic value becomes normal after surgery

Conclusions

The diagnosis of coarctation of the aorta is aided by the study of com parative pulse and pressure tracings between the upper and the lower extremities The electrokymograms of the aorta may demonstrate the exact area of the narrow section

AORTITIS ATHEROSCLEROSIS OF AORTA

These two processes have been classified in the past under the same name of aortitis' It is preferable to keep them apart because of their different etiologies and manifestations However inasmuch as the two forms can be associated a clinical differentiation is not always possible

Aortitis is due to an inflammatory lesion usually syphilitic It is usually located in a limited portion of the vessel and in one or few of its branches the ascending aorta and the aortic arch being the preferred sites

Atherosclerosis of the aorta may occur in young people but, as a rule is found after 50 and is frequent in old age The atherosclerotic aorta is longer

* Exceptional cases have a lower pressure and a smaller pulse in the left brachial artery on account of constriction of the left subclavian artery

and larger than a normal vessel The process is most frequent in the abdominal aorta

Aortitis or Atherosclerosis of the Ascending Aorta and Aortic Arch

This localization is frequently accompanied by coronary arteriosclerosis aortic insufficiency, or both

ELECTROCARDIOGRAM *This tracing may present different patterns caused by* associated lesions Thus changes due to aortic insufficiency (p 336) hyper tension (p 476), bundle branch or intraventricular block (pp 429 and 436) and coronary heart disease are frequently encountered There is no relative bradycardia like in aortic stenosis (p 346)

PHONOCARDIOGRAM The phonocardiogram may reveal the following data

1 An *opening click* (or snap) of the aortic valve this is a high and loud vibration which precedes the rise of the carotid pulse by from 0 01 to 0 02 second and is due to lesion of the semilunar valves This vibration is a part of the first sound complex and is larger than the other vibration occurring at the time of a v valve closure (p 42)

2 A triple rhythm due to addition of a loud systolic vibration (so called *systolic gallop*) [4] the first sound complex is followed by two or more low pitched high vibrations which are simultaneous with the systolic ex pansion of the aorta (peak of the carotid wave)

3 The second sound consists of three to four large vibrations and is prolonged by two or three smaller vibrations equivalent to a short diastolic murmur

4 A *systolic murmur* is also frequently present its vibrations have either the aspect of a short early systolic murmur or that of a diamond shaped murmur It is recorded best over the second or third right interspaces but may be loudest at the left of the sternum

In cases of syphilitic aortitis the fine high pitched vibrations of friction rubs may be recorded near the manubrium

LOW FREQUENCY TRACING (CARDIOGRAM) The apex cardiogram is of normal aspect or reveals a rapid high pulsation There is no slow staggered rise like in aortic stenosis (p 346)

PULSE AND PRESSURE TRACINGS The pulse tracings reveal a steep rise a sharp peak and a quick descent These changes may be due to either aortic insufficiency (syphilis) or increased rigidity *of the aortic wall (atheroscler osis)* There is no anacrotic depression like in aortic stenosis The pulse and pressure tracings of the lower extremities frequently reveal an increased pres sure Atherosclerosis of the aortic arch may cause smaller pulse and lower blood pressure in the arms (narrowing of the subclavian arteries) The resulting picture is the opposite of that of coarctation (p 466)

Electrokymogram Both the border tracings and the densograms of the ascending aorta and the aortic arch have pulsations of normal or increased magnitude The pulses have a steep rise, a sharp peak and a rapid descent There is no anacrotism in the descending limb These data like those of the pulse tracings are useful in the differential diagnosis with aortic stenosis

Atherosclerosis of the Abdominal Aorta

Pulse Tracing, Phonoarteriogram The pulse tracing of the femoral arteries has a great amplitude Its pattern consists of a sharp rise and a rapid drop

Simultaneous sound tracing over the second right interspace and femoral pulse tracing (or simultaneous pulse tracings of the suprasternal notch and the femoral arteries) reveal an increased speed of the pulse in the descending aorta The femoral pulse normally is simultaneous with the brachial in these patients it may precede the brachial pulse (Fig 255)

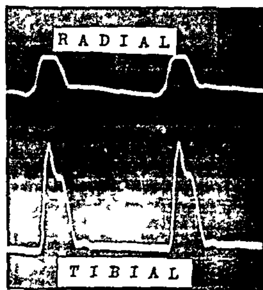


Fig 255 Pulse tracings of a patient with atherosclerosis of the abdominal aorta

A systolic murmur is frequently recorded over the abdominal aorta

CONCLUSIONS

Graphic data of diagnostic value are

1 A diamond shaped aortic systolic murmur (phono)
2 A large pulse with rapid rise and rapid fall (carotid tracing aortic eky)
3 An apex cardiogram with rapid high pulsation

These data together with the electrocardiogram (moderate or absent left ventricular hypertrophy no bradycardia) are sufficient for a correct diagnosis and for excluding aortic stenosis which might cause a similar systolic murmur

The diagnosis of atherosclerosis of the abdominal aorta is helped by the study of the aortogram

ANEURYSMS

The great majority of the aneurysms of the aortic arch are syphilitic while most of those of the abdominal aorta are caused by an atherosclerotic lesion The aneurysm may be fusiform or saccular The blood contained in the pouch

may remain fluid in such a case the aneurysm acts like a reservoir which dilates during systole and shrinks with diastole In many cases however stratified thrombi fill the pouch and this behaves like a solid mass Com pression of one or more arteries is possible and may lead to unilateral or bilateral disappearance of the radial pulse

When the aneurysm grows outward it forms a mass which pulsates with an expansive pulsation In certain cases the systolic thrust of the apex is followed by a diastolic thrust due to the recoil of a retrocardiac aneurysm (double pulsation) Auscultation of the mass reveals the heart sounds and a double murmur caused by the blood entering and leaving the pouch through a narrow opening

Electrocardiogram

Possible changes are those already mentioned in cases of aortitis and atherosclerosis of the aorta

Pulse Tracings Cardiogram

Severe changes of circulatory dynamics take place when the aneurysmal sac is large elastic and contains fluid blood if the opening is narrow the effects are more marked The sac behaves like a reservoir because it fills during systole decreasing the systolic pressure below it and gives back the blood in diastole increasing the diastolic pressure below it Therefore a comparison between an artery above and one below the sac reveals that the pulse is smaller and has a slower rise (*pulsus tardus*) in the arteries placed below

In *aneurysm of the ascending aorta* a high but slowly developing pulsation is recorded over the second and third right interspaces where the pouch con tacts the soft parts of the anterior chest wall The apex cardiogram may reveal a double thrust (one in systole one in diastole) or only a diastolic thrust due to the rebound of the aneurysm which displaces the entire heart The tracings of the brachial arteries reveal a simultaneous rise of the two pulses however the right pulse is smaller than the left and has a slower rise so that its peak is delayed (Fig 256)

In *aneurysm of the aortic arch* a typical high and slow pulsation is recorded at the suprasternal notch The brachial pulses have a simultaneous rise but the left pulse is smaller and has a slower ascending branch so that its peak is delayed (Fig 257)

The *aneurysm of the innominate trunk* causes a smaller and delayed pulse in the right arm while that of the left subclavian artery is followed by similar changes in the left arm (Fig 258)

The *aneurysm of the abdominal aorta* is revealed by a high and slow pulsation over the pulsating mass The femoral pulse is smaller and has a slower rise in comparison with the brachial pulse (Fig 259) Two si

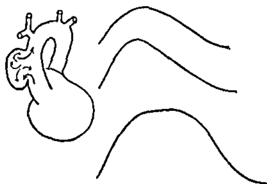


Fig 256 Scheme of the pulses in the aneurysm of ascending aorta From above right radial left radial and tibial pulse

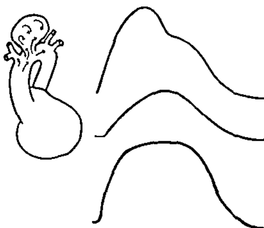


Fig 257 Scheme of the pulses in the aneurysm of aortic arch (see Fig 256)

multaneous aortograms recorded at the two opposite sides of the mass may aid in the differential diagnosis with a tumor an aneurysm has an expansive pulsation revealed by two positive pulses, a tumor has a transmitted pulsation and this is revealed by a positive and a negative pulse

Phonocardiogram Phonoarteriogram

Various changes caused by myocardial damage (p 418) or aortic valvular lesion (p 336) may be observed

Records taken over the pulsating aneurysm (ascending aorta or aortic arch) reveal a systolic and frequently also a diastolic murmur It is difficult to say whether these are caused by the blood moving in and out of the pouch or are transmitted from the aortic valve In such a case dilatation of the ascending aorta (relative stenosis of the ostium) would cause the first, valvular lesion (aortic insufficiency), the second

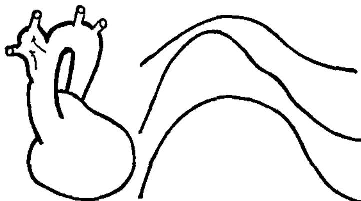


Fig 258 Scheme of the pulses in the aneurysm of innominate trunk (see Fig 256)

In aneurysm of the abdominal aorta, a tracing recorded over the pouch reveals a rough systolic murmur seldom also a diastolic

Electrokymogram

Two types of tracings can be taken border tracings and densograms of the pulsating mass

A border tracing presents a typical pattern the waves are large and have a slow rise and a slow fall in any case where the pouch is expanding and contains fluid blood On the contrary, the pattern is identical to that of the aortic pulse when the pouch is full of clots This tracing cannot be used for differential diagnosis with tumors because both an aneurysm and a tumor may present border pulsations of arterial type due to mechanical transmission of the aortic pulse

The densogram of an aneurysm has a large pulsation with a slow curve of the pulse This can be easily differentiated from the densogram of a tumor which having a poor blood supply presents a small pulsation or none at all (Fig 260) [3, 6, 8] When a tumor is over the aorta or the pulmonary artery, the most suitable projection should be selected in order not to record the pulsation of these vessels

In aneurysm of the pulmonary artery, typical changes are observed, not only over the main trunk but usually also over one or both stems Thus the hilar shadows frequently present large and slow pulse waves with the pattern found in aneurysms

DISSECTING ANEURYSM OF THE AORTA

Different types of tracings can be obtained according to the severity and extension of the dissection In simple tear of the ascending aorta a soft, early diastolic murmur is recorded by the phonocardiograph over the second right interspace This is due to lack of support of one of the aortic cusps causing aortic regurgitation

If there is compression of the coronary arteries a typical pattern of ischemia or injury may be revealed by the electrocardiogram

If the dissection progresses and there is compression of one of the brachial arteries the pulse tracing of the latter shows small and possibly delayed waves

In dissecting aneurysm of the abdominal aorta a pulsating mass becomes apparent in the abdomen The femoral pulse is small and delayed or disappears The extremely small amplitude of the pulse is a differential sign with other types of aneurysm of the abdominal aorta

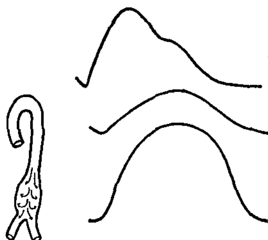


FIG 259 Scheme of the pulses in the aneurysm of the abdominal aorta (see Fig 256)

CONCLUSIONS

The diagnosis of aneurysm of the aorta and its exact location are considerably aided by the study of the various pulse tracings and by the electrokymogram Furthermore the electrokymogram gives data which are of help in the differential diagnosis between aneurysms and tumors

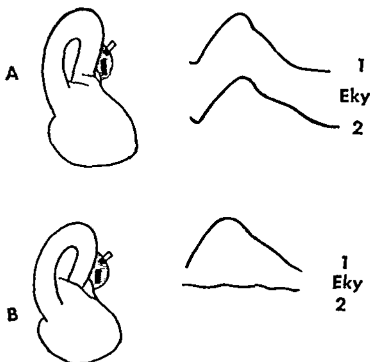


FIG 260 Scheme of the electrokymogram in aneurysm of the thoracic aorta (*A*) and in mediastinal tumors (*B*) *1* Border tracing *2* densogram

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CHAPTER 52

Hypertension

SYSTEMIC HYPERTENSION AND HYPERTENSIVE HEART DISEASE

Arterial hypertension is frequently a secondary occurrence in diseases of the kidneys or the arteries It may be present as a solitary occurrence Hypertensive heart disease is not the same as hypertension , such term should be used only when there is evidence of left ventricular hypertrophy and ischemia

Electrocardiogram

The early stages of hypertension may not be accompanied by electrocardiographic changes However, a short P R interval (about 0 14 second) with *normal QRS has been described* [12]

The electrocardiographic evidence of left ventricular hypertrophy is considered one of the most important signs of hypertensive heart disease (p 418) The unipolar limb leads may show an inverted T wave either in aVL (left arm) or in aVF (left leg) according to the position of the heart For this reason while such pattern is seen most commonly in lead 1 it may be present occasionally also in lead 3 [5]

Patients with renal hypertension may also present low voltage of P and QRS and prolongation of Q T This electrocardiographic picture has been called *cor renale* [16]

Phonocardiogram

This tracing may show high and prolonged vibrations caused by a loud booming second aortic sound the vibrations of the first aortic sound are often prolonged

A systolic murmur is frequently recorded at the apex over the aortic area or both The former is due to left ventricular enlargement causing slight mitral regurgitation and may be favored by fibrosis of the mitral valve The latter is caused by enlargement of the left ventricle and the ascending aorta resulting in a relative aortic stenosis (p 351)

It is common to record a triple rhythm The meaning and mechanism of this have been already discussed (p 422)

Pulse and Pressure Tracings

The pulse curve may present an anacrotic depression indicating left ventricular strain There may be pulsus alternans evidence of a severe functional abnormality of the left ventricle (p 428)

The pressure tracing recorded with both the oscillatory and auscultatory methods (p 154) frequently reveals an *auscultatory gap* This consists of a lack of arterial sounds while the pulses are unchanged the phenomenon seems connected with the anacrotism of the pulse (p 350)

The conventional limit of the upper range of blood pressure is 150/90 Whenever a temporary increase of blood pressure can be excluded a systolic pressure higher than 150 mm or a diastolic pressure higher than 90 mm should be considered abnormal In hypertension systolic pressure may reach the extreme level of 300 mm of mercury diastolic pressure is usually also elevated In the author s experience diastolic pressure is much higher in secondary hypertension of nephritis and in malignant hypertension than in essential hypertension therefore pulse pressure is usually greater in the latter

Different readings in the two arms with a higher blood pressure in either the right or the left arm (more often the left) are common The systolic pressure of the lower extremities is usually from 30 to 40 mm higher while the diastolic pressure is from 10 to 20 mm higher than that of the arm A greater difference points to either aortic insufficiency or abdominal aortitis The opposite behavior namely lower pressure in the legs is a sign of coarctation of the aorta High pressure in the legs with low pressure in the arms may be caused by hypertension plus atherosclerosis of the aortic arch (p 469)

It has been said that an isolated hypertension of the lower extremities may precede by years the establishment of hypertension in the higher limbs This fact however needs confirmation

Venous Pressure

Venous pressure may be normal or low However, cases of essential hyper tension where venous pressure reached 14 and even 16 cm H O without evidence of heart failure have been described This elevation has been at tributed to partial transmission of the high arterial pressure

Cardiomanometry

Catheterization has shown that normal pressures are found in both the right ventricle and the pulmonary artery in cases with benign hypertension On the other hand elevated pressures have been recorded in malignant hypertension as well as in cases with heart failure

Peripheral Circulation

Radial pulse tracings should be correlated with digital plethysmograms In hypertension the following changes were observed by Dillon and Herzman [3]

1 Increased crest time (p 139) in both the digital and radial pulses
2 Disappearance of the dicrotic wave in both tracings
3 Triangular shape of the digital pulse, rounding and flattening of the radial pulse

The increase of the crest time has been explained with the appearance of a small wave between peak of the main wave and incisura This wave is absent or very small in normal subjects and seems to be related to an increase of peripheral resistances

These changes are similar to those caused by arteriosclerosis (p 484) and become significant if noted in young or middle aged individuals

Electrokymogram

The border tracing of the left ventricle may present several abnormalities which indicate suffering and strain of this chamber Apart from the result of bundle branch or intraventricular block and from the occurrence of alternans or premature contractions, the following data may be observed

1 Increase of the isometric relaxation period [2]
2 High atrial wave
3 Delayed onset of contraction
4 Small initial drop followed by a more rapid drop in the second part of systole
5 Severe drop, or deep rebound in early diastole

In addition the changes due to a dynamic aneurysm (p 453) can be observed they are evidence of single or multiple old myocardial infarcts

The border tracing of the aortic arch may reveal changes similar to those of the arterial tracing e g anacrotic depression alternans or the irregular waves due to premature contractions

Ballistocardiogram

According to Starr and Jonas[14] the ballistocardiogram of hypertensive patients frequently reveals low cardiac output and an abnormal pattern The findings are similar to those encountered in coronary heart disease or arteriosclerosis (p 484) Therefore they have a certain value only if present in young individuals without evidence of coronary heart disease

Conclusions

Pulse and pressure tracings indicate whether or not there is arterial hyper tension and whether this is diffused to the entire system or limited to the higher extremities like in coarctation of the aorta They may reveal the existence of anacrotism auscultatory gap or alternans evidence of abnormal left ventricular contraction

The electrocardiogram reveals whether or not the left ventricle is hyper trophied and has ischemia of the wall in such a case hypertensive heart disease should be admitted It further shows whether increased excitability (premature contractions) or myocardial damage (intraventricular block bundle branch block old infarcts, etc) is present

The phonocardiogram may reveal evidence of left ventricular strain (triple rhythm functional murmurs)

The electrokymogram may reveal several data indicating abnormal left ventricular contraction including prolongation of the isometric relaxation period and slow late or irregular descending branch of the ventricular wave

An abnormal ballistocardiogram may confirm the strain and suffering of the left ventricle and reveal a low cardiac output

HYPERTENSION OF THE LESSER CIRCULATION AND CHRONIC COR PULMONALE

The syndromes caused by hypertension of the lesser circulation have been described more recently than those caused by systemic hypertension A secondary lesion of the right heart is a frequent, though not inevitable, result

Chronic pulmonary heart disease is the result of persistent hypertension of the lesser circulation Even if this hypertension is frequent in patients with mitral stenosis or congenital lesions of the heart the term 'pulmonary heart disease should be restricted to cases where the high pressure of the pulmonary artery is due to extracardiac causes

Several types of chronic cor pulmonale have been recognized

1 That of patients having idiopathic pulmonary hypertension or arterio sclerosis of the pulmonary vessels Increased resistance in the arterioles causes an increased work of the right ventricle There is little or no impairment to oxygen intake in the pulmonary capillaries

2 That of patients with pulmonary emphysema or pulmonary fibrosis

resulting from chronic inflammation of the lungs (tuberculosis, abscess bronchiectasis, chronic bronchitis, bronchial asthma) An obstruction of the capillaries increasing the load of the right ventricle, is present in these patients Hypoxia resulting from the primary pulmonary lesion complicates the picture

3 That of patients with chest deformities Here, impairment of respiration is due to abnormal chest structure and abnormal respiratory dynamics

4 That of patients with a v fistula of the lung where the shunt prevents oxygenation of a certain percentage of the blood

Electrocardiogram

Cardiac rhythm is usually regular, cardiac rate is nearly always increased

Right axis deviation is frequent Right ventricular hypertrophy is usually noted (p 420) and there may be evidence of ischemia of the right ventricular wall depressed S T and inverted T wave in leads 2, 3, and aVF, as well as in the chest leads V1-V2 There may be a *P pulmonale,* which is evidence of right atrial enlargement (p *421*) *This consists of high voltage and longer duration* of P in leads 2 3, and aVF There is a positive phase of P, followed by a brief and peaked negative phase in V1-V2, the duration of the diphasic P is greater than 0 10 second (Fig 222)

Phonocardiogram

A diamond shaped systolic murmur is often recorded over the second left interspace It is caused by the dilatation of the pulmonary artery (relative stenosis) The second pulmonic sound is loud and occasionally split It may be followed by a few vibrations in decrescendo caused by slight incompetence of the pulmonic valve due to either high pressure (functional pulmonic in sufficiency) or valvular fibrosis (organic pulmonic insufficiency) In an advanced stage it is possible to record the vibrations of a systolic murmur over the tricuspid area (functional tricuspid insufficiency)

A triple rhythm is frequently recorded (p 422) over the epigastrium and the tricuspid area (so-called right ventricular gallop)

The murmurs of the a v fistula of the lung are the following [11] A late systolic murmur frequently overshadowing the second sound or an early diastolic murmur

These murmurs are louder during inspiration[11] and are recorded in the area which is nearest the fistula

Low Frequency Vibrations of the Chest and Epigastrium

An apex cardiogram may reveal a systolic depression of the apex On the contrary the epigastric tracing usually reveals a high early and forceful positive thrust due to the hypertrophy of the right ventricle (p 422) The atrial wave is high in the epigastric tracing [17]

Jugular and Hepatic Tracings

Both tracings reveal a high presystolic atrial wave In advanced stages relative tricuspid insufficiency may be revealed by a typical plateau like positive wave in systole (p 359)

Electrokymogram

The tracing of the right ventricle may indicate the severity of myocardial damage through abnormality of the contraction pattern

Usually the most notable findings are the following [7, 8, 9]*

1 High and rapidly expanding pulse of the pulmonary arch with a high dicrotic wave
2 High and rapidly expanding pulse over the hilar shadows
3 Small or absent pulsation over the lung fields In cases with unilateral or lobar fibrosis this absence of pulsation is limited to the affected side or to the fibrotic lobes

In cases with a v fistula of the lung border tracings and densograms of the shadow reveal a high and rapid pulsation of an arterial type [11]

Cardiomanometry

Right ventricular systolic pressure is between 34 and 90 mm mercury (normal 22–23) right ventricular diastolic pressure is frequently 3–5 mm mercury (normal 0) therefore there is an increased pulse pressure If the right ventricle is in failure its diastolic pressure becomes higher The systolic pressure of the pulmonary artery is about the same as that of the right ventricle e g 34–90 (normal 22–23) while the diastolic is frequently 10–30 (normal 7–9) [1, 4, 6, 10]

The gradient of pressure between pulmonary artery and pulmonary capillaries is frequently increased indicating pulmonary arteriolar vasoconstriction Oxygen inhalation or intravenous digitalis frequently reduces this gradient indicating its functional nature If the patient is in failure the pressures of the right ventricle and pulmonary artery are remarkably higher and that of the right atrium is above zero during all phases of the cardiac cycle

In cases with a v fistula of the lungs the pulmonary artery has a high systolic and a low diastolic pressure The oxygen saturation of the right heart as well as that of the arterial blood is frequently low in chronic cor pulmonale It is particularly low in cases with a v fistula of the lungs

* The frequently existing dyspn a makes the takin of a record in apnea difficult Therefore use of a special electric filter and study during normal re piration may become necessary [8]

Respiratory Tracings

Tracings of respiration may indicate the existence of an important broncho spastic component, revealed by prolonged duration of expiration This would undoubtedly increase the severity of dyspnea

Spirograms may indicate whether the vital capacity is decreased

Conclusions

The electrocardiogram gives data indicating right ventricular hypertrophy and ischemia and right atrial enlargement

The phonocardiogram reveals the effects of high pressure in the pulmonary artery (systolic murmur loud P2) and may reveal an initial valvular in sufficiency (early diastolic murmur) It frequently shows evidence of right ventricular strain (triple rhythm)

The low frequency tracing gives evidence of right ventricular enlargement (high systolic pulsation at the epigastrium) while both this and the venous tracing reveal right atrial enlargement (high atrial wave)

Evidence of obstruction in the arterioles or capillaries of the lungs is supplied by the electrokymograph which reveals a contrast between the large arterial vessels (high pulsation) and the pulmonary parenchyma (small or no pulsation)

Catheterization of the heart indicates exactly the degree of hypertension of the pulmonary artery and whether there is functional vasoconstriction (high reversible gradient between pulmonary arterial pressure and capillary pressure), it may reveal right ventricular failure (high diastolic pressure of the right ventricle)

Respiratory tracings may reveal the part played by bronchial and pulmonary elements in causing dyspnea

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CHAPTER 53

Peripheral Vascular Diseases

Peripheral vascular diseases may involve the arterial system the venous system, or both

Arterial vascular disorders can be divided into functional and organic vascular conditions The best known functional disorders are Raynaud s disease, acrocyanosis, erythromelalgia and the vasospastic states caused by prolonged exposure to extremes of temperature The most common organic arterial diseases are arteriosclerosis obliterans, periarteritis nodosa arterial thrombosis and embolism

Venous diseases include thrombophlebitis and varicose veins Arterio venous fistulas and Buerger s disease involve mostly the medium sized arteries (ulnar, radial, posterior tibial and dorsalis pedis) and only later the larger vessels Arteriosclerosis obliterans involves the larger arteries (femoral popliteal iliac) and only later, the posterior and anterior tibial arteries Usually, it does not involve the vessels of the upper extremities Both Buerger s disease and arteriosclerosis obliterans are revealed by intermittent claudica tion

PULSE AND BLOOD PRESSURE TRACINGS

These tracings reveal the level of pressure in the various arteries the amplitude of pulsations, and the configuration of the pulses

The most varied findings can be obtained In certain cases no pulse tracing (and no indirect pressure tracing) can be obtained over certain arteries This is usually due to occlusion of the vessels However extreme narrowing of an artery may still allow a thin continuous flow of blood which cannot be recognized by these tracings on account of the lack of pulsation A similar phenomenon is observed in coarctation of the aorta (lower extremities only), and in aortic stenosis shock or congestive failure (all arteries) Absence of pulsations below a given point is suggestive of arterial occlusion in cases of embolism

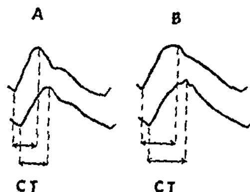


FIG 261 Radial and digital pulses *A* normal subject *B* patient with peripheral arteriosclerosis (*CT* crest time)

Senile arteriosclerosis is accompanied by changes in the configuration of both the radial and the digital pulse In patients with this disease crest time (p 139) is markedly increased in both the radial and the digital tracings (Fig 261) Younger persons with initial arteriosclerosis may present an increase of the crest time of the digital pulse *It has been also noted that in arteriosclerosis the dicrotic waves decrease sharply* and may disappear in the tracings of the fingers and toes [7]

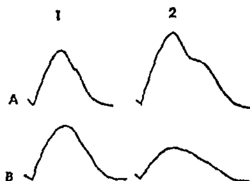


FIG 262 Changes of the arterial pulse of the lower extremities before exertion (*1*) and after exertion (*2*) *A* normal subject increased amplitude of the pulse *B* patient with arteriosclerosis obliterans decreased amplitude of the pulse

Pulse and pressure tracings can be recorded before and after exercise on a special bicycle In normal subjects increased blood pressure and larger pulses are observed after exertion On the contrary patients with organic arterial disease with or without intermittent claudication present an inverse reaction with decrease of blood pressure and smaller pulses in the lower extremities[9] (Fig 262)

In arteriovenous fistula a normal or low systolic and a low diastolic pressure are observed Pulse pressure is markedly increased, the pulse wave has an increased velocity The predicrotic notch may be at a low level [8, 10] The arterial pulse may be weak and even disappear below the fistula

VENOUS PRESSURE TRACING

In arteriovenous fistula local venous pressure is elevated while systemic venous pressure is normal

PHONOCARDIOGRAM

A sound tracing recorded over the abdominal aorta frequently reveals a systolic murmur in peripheral vascular diseases[11] This murmur is probably due to the association of atherosclerosis of the abdominal aorta with obstruction of flow in the iliac or femoral arteries

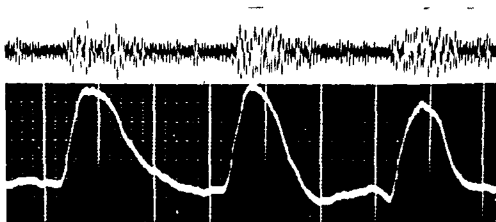


FIG 263 Phonoarteriogram and pulse tracing of a traumatic arteriovenous fistula at the groin Continuous murmur with systolic increase

In arteriovenous fistula sound tracings reveal a continuous murmur with vibrations of both high and low pitch The vibrations are larger during the expansion of the artery smaller during its collapse (Fig 263) The two phases coincide with ventricular systole and diastole if the fistula is near the heart while they are delayed over such phases in more peripheral vessels

The sound tracing has been used for the differential diagnosis with an arterial aneurysm compressing the veins Such an aneurysm usually presents no audible diastolic murmur However the sound tracing may reveal minute diastolic vibrations, especially if there is aortic regurgitation Whenever the fistula is within the chest (aorta to superior cava) the murmurs increase in inspiration[9]

PLETHYSMOGRAPHY

Maximal blood flow in the most affected limb of patients with Buerger's disease was found decreased by Kunkel and Stead within normal limits by

Landowne[6] and by Abramson [1] This contrasted with the clinical symptoms of exertional pain and claudication Functional tests seem on the other hand to reveal the existence and severity of arterial narrowing [8]

Maximal blood flow was found severely decreased in arteriosclerosis obliterans by Kunkel and Stead[5] but symptoms and trophic changes were absent unless the reduction was below 33 per cent

BALLISTOCARDIOGRAM

The ballistocardiogram of arteriosclerotic patients frequently presents abnormal patterns [1] These are extremely variable and are similar to those already described in coronary heart disease (p 456) They may have importance if found in relatively young persons (Fig 264)

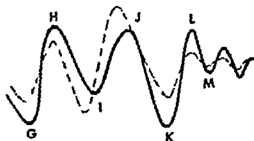


Fig 264 Ballistocardiogram of arteriosclerosis The dotted line indicates the normal pattern

CONCLUSIONS

Pulse tracings blood pressure tracings plethysmograms and ballistocardiograms yield important data in peripheral vascular diseases Arterial and venous pressure readings sound tracings and functional tests also contribute to diagnosis in some of the diseases

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CHAPTER 54

Heart Failure

RESPIRATORY TRACINGS

Patients with paroxysmal dyspnea present increased respiratory rate There frequently is a *prolonged expiratory phase*[6, 7] (cases with wheezing respiration) similar to that observed in bronchial asthma (Fig 265) Both cardiac and allergic patients fail to reveal a shortening of expiration after exercise like normal subjects and both reveal a shortening of expiration during intravenous administration of aminophyllin[6] These phenomena are probably caused by reflex bronchospasm following pulmonary congestion

Cheyne Stokes respiration may occur in certain cases during paroxysmal dyspnea However this type of respiration may be observed also in chronic congestive failure Two varieties have been described (Fig 266) that with gradual transition between phases of apnea and phases of dyspnea and that with sudden change from one phase to the other

In *exertional* and in *continuous dyspnea* of a cardiac patient the tracing reveals an increased rate and, in some cases, an oblique course of the inspiration[7]

ELECTROCARDIOGRAM

This tracing reveals various disturbances of the cardiac rate and rhythm which are indirectly connected with congestive failure and dilatation of the

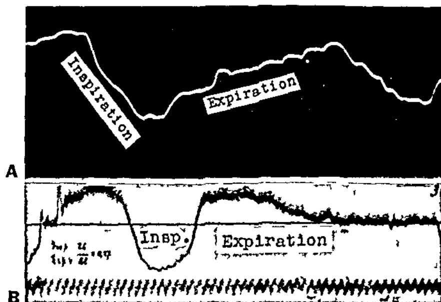


FIG 265 (A) Thoracic tracing in paroxysmal dyspnea increased duration of expiration (B) Pneumotachogram in a case of allergic asthma Extreme increase of expiration

heart The most frequent are sinus tachycardia, atrial flutter or fibrillation, ventricular premature contractions and ventricular tachycardia

Two abnormalities have been attributed to abnormal metabolism of the myocardium causing failure (so called energeto dynamic type of failure)
1 Low voltage and increased duration of QRS, prolonged P R interval [1]
2 Prolongation of Q T [8]

The latter may cause dissociation between electrical and mechanical systole so that the end of T falls after the second sound

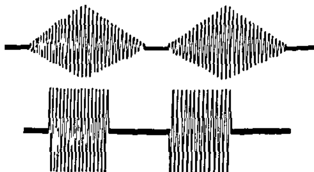


FIG 266 The two varieties of Cheyne Stokes respiration

JUGULAR TRACING

Congestive failure is accompanied by a progressive engorgement of the veins revealed by progressive flattening and early end of the systolic collapse [9] This may be favored by relative tricuspid insufficiency (p 359)

CARDIOMANOMETRY

Left Ventricular Failure

Failure of the left heart causes increase of the residual blood in the left ventricle and atrium which is followed by increased diastolic pressure of the left ventricle and increased left atrial pressure High pulmonary artery pressures frequently follow but the pressure gradient between pulmonary artery and pulmonary "capillaries is unchanged Intravenous digoxin increases the output of the left ventricle and decreases systolic and diastolic pressures of the pulmonary artery [4]

Right Ventricular Failure

Patients with right ventricular failure caused by increased resistance in the circulation of the lungs (cor pulmonale) present increased diastolic pressure in the right ventricle and the right atrium The gradient of pressure between pulmonary artery and pulmonary capillaries is frequently increased probably on account of pulmonary vasoconstriction apparently due to anoxia Intravenous digoxin increases right ventricular and pulmonary systolic pressures and right ventricular output it decreases right ventricular and pulmonary diastolic pressures the pulmonary hypertension caused by the drug is only temporary and decreases subsequently [5] so that the pulmonary arterial pressure may become stabilized at a normal level

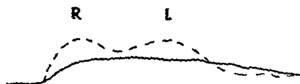


FIG 267 Radiocardiogram in congestive failure Prolongation and fusion of the waves *R* and *L* indicating passing of the radioactive material through the two ventricles The dotted line indicates the normal pattern (after Prinzmetal)

RADIOCARDIOGRAM

Patients in congestive failure have a slow rise of the first wave (R wave) incomplete or complete fusion of this with the second wave (L wave) severe prolongation of the L wave (Fig 267) [10] These changes are due to incomplete emptying of the ventricles with persistence of the radioactive material in either of the cardiac chambers or both

VENOUS PRESSURE TRACINGS

Venous pressure may be increased by either obstacle to circulation (tricuspid stenosis) or heart failure Increased venous pressure of heart failure is

thought to be due to several factors (including venous hypertonus) which are only indirectly related to the weakness of the heart Still, repeated tracings may reveal whether the failure is increasing or decreasing because the venous pressure level varies inversely with the efficiency of the myocardium

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APPENDIX

Descriptions of Various Apparatus

ELECTROCARDIOGRAPHS

The action potentials spreading from the heart are in the order of magnitude of fractions of millivolts (mv) and present rapid oscillations A suitable recording apparatus must be sensitive rapid and exact, so that the various waves are recorded without delay or distortion This can be done (1) by employing a string galvanometer of high sensitivity or (2) by amplifying the action potentials so that they can be recorded by an electromagnetic galvanometer In both cases the galvanometer should have a high frequency period in order to record correctly the rapid oscillations and should be properly damped in order to return promptly to the initial position

String Electrocardiograph

The electric currents of the heart are recorded without amplification by means of *Einthoven s string galvanometer* This consists of a thin metallic coated quartz string through which flow the currents The string moves within the magnetic field of an electromagnet according to Ampere s law The shadow of the string is projected onto the film by a system of magnifying lenses the movement of the shadow combined with the linear movement of the paper, creates a graph or tracing The extremely small mass of the string insures a high recording fidelity however the sensitivity of the system can be regulated only by modifying the tension of the string A current of 1 mv switched from a dry cell into the circuit which includes the patient is used for standardization a deflection of 1 cm should be recorded

The current which flows through the string is directly proportional to the voltage and inversely proportional to the resistance of the circuit mostly caused by skin resistance therefore the electrodes should be carefully and properly applied

Inclusion of condensers in the circuit serves to exclude currents produced by the various structures of the body while a resistance compensates for possible polarization currents of the electrodes The spokes of a turning wheel are used for marking time lines on the film

These basic principles of construction of Einthoven s electrocardiograph are still applied in the construction of certain modern apparatus which however are operated by alternating current In one of these apparatus the *Cambridge All Electric Electrocardiograph* only the standardization current is furnished by a battery supplied with an adjustment rheostat This current can be reversed if the string

when compensating cannot be brought back to the center of the field A *lead control knob* connects the string to the various leads (1,2 3), while in a fourth position, a resistance of 2000 ohms is connected in series with the string * An automatic induction eliminator for AC can be used whenever the typical AC interference (or hum) occurs revealed by a fuzziness of the string shadow

The *Portable All Electric Cambridge Apparatus* is contained in two cases the larger contains galvanometer, lamp camera, time marker, and control panel the smaller contains power unit and compartments for accessories

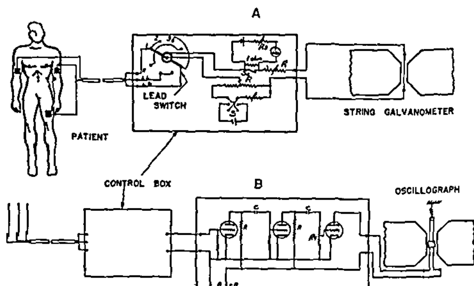


FIG 268 *A*, Circuit of a string galvanometer *B*, Circuit of an oscillograph with thermionic amplifier (From *Medical Physics*, courtesy of Year Book Inc)

A more modern machine is the *Cambridge Simpli Trol* It is of smaller size and is housed in a single case Standardization of the galvanometer is accomplished by a two way switch providing potentials of 1 and 3 mv in order to check the proportion of string deflections

Amplifying Electrocardiograph

In this type of instrument amplification of the cardiac potentials is accomplished by thermionic vacuum tubes In the thermionic vacuum tube a current of electrons goes from the heated cathode toward the plate if a difference of potential occurs

* To determine the patient s resistance the following formula is suggested by the Cambridge Co $Rp=A\ (Rs+2000)=Rs$

A=string deflection in cm when lead knob is in fourth position

Rs=string resistance Rp=patient resistance

The operation is as follows with the patient connected and the lead control knob placed on 1 standardize the string deflection to 1 cm/mv Then without changing the tension of the string switch to position 4 deflection A will be found when 1 mv is placed in the circuit In general if deflection A does not exceed 1 3 cm the patient resistance is within average limits

between them An interposed grid, with low negative potential controls the flow of current in the plate circuit and a proportional variation of voltage takes place across the resistor placed in the plate circuit Thus variation of voltage across the plate resistor is similar to that of grid voltage even though amplified

In the amplifying electrocardiograph the patient is connected with the grid circuit of a multistage thermionic amplifier the plate circuit of each tube is closed by some form of impedance and the changes of potential manifested across the impedance load are then transmitted to the grid of the next tube The grid and plate resistors are linked by a condenser which acts as an isolating medium for the direct current potentials of the grid and plate circuits if the changes of potential are of a pulsating character, they pass through the condenser A suitable degree of amplification can be obtained with a three stage system The degree of amplification provided by the thermionic vacuum tubes can be modified by the resistance-capacity circuit itself For correct recording of the low frequency waves the time required by the oscillograph to reach one half a previous standard deviation should be 1.5 seconds This property of the circuit is known as the *time constant* and is one of the most important characteristics of the electrocardiograph *

The amplified potentials are recorded by an oscillograph Essentially, the oscillograph consists of a loop (or coil) of wire which moves within the field of a permanent magnet when currents flow through it Attached to the loop is a small mirror which throws a beam of light In other types a small piece of metal carries the mirror and moves within the field of an electromagnet. The frequency of an oscillograph must be high enough to record rapid variations of potential (200 to 400 Hertz), the minimum time required for a deflection is usually 0 01 second The oscillograph must be damped so that it can return to the position of rest soon after a deflection

Many types of amplifying electrocardiographs are now available some operated by batteries others by AC Most of them are portable therefore both the sturdiness of the galvanometer and its limited weight are important qualities

In the *Sanborn Instomatic Cardiette* the beam of light received by the mirror is crossed by a line, therefore the resulting tracing is white-on black' Modern apparatus have devices for lead selection (1,2 3 V) and for checking batteries they also incorporate a device for automatic compensation between leads permitting the rapid switch from one to the other (*instomatic*)

Cathode Ray Electrocardiograph

In this type of oscillograph a beam of cathode rays is deviated by the electrostatic deflection plates which are directly connected with the output of the amplifier Theoretically it is the most perfect oscillograph because it has a uniform frequency response and no inertia the deviations of the spot of light being photographed by a uniform speed camera While practical advantages for clinical electrocardiography are not significant, the cathode ray tube is used for visualization of the

* This property of automatic return to the baseline after a deviation explains why a compensating circuit is not needed in the amplifying electrocardiograph On the other hand this instrument is not suited for recording extremely slow changes of potential

electrocardiogram on a fluorescent screen (*oscilloscope*) and in the construction of the *vectorcardiograph*

Modern apparatus frequently include in the same assembly two or more systems of amplification in order to record simultaneously two or more electrocardiograms or an electrocardiogram and a tracing of heart sounds (stethogram or phonocar diogram)

Direct Writing Electrocardiographs

These electrocardiographs are operated by AC The most important advantage of these instruments consists of the fact that the tracing can be read at once These cardiographs are of the amplifying type great power, delivered by the amplifier to the galvanometer is used in order to move a *writing arm* or *stylus* A heated stylus writing on heat sensitive paper is used in the best modern apparatus while others write by means of chemical devices or ink Since the writing stylus describes an arch when moving different devices are used in order to obtain accurate rectangular coordinates in the tracing In the *Cambridge Simpli Scribe*, the film moves within a concave groove having the same radius as the writing stylus In the *Sanborn Viso Cardiette* the film is pulled over a sharp edge and the heated stylus swings over this edge thus producing records with true rectangular coordinates and a negligible tangential error (0 5 mm in the extreme positions of the stylus)

The *Sanborn Poly Viso Cardiette* has a wide potential range of use in clinical and physiologic research It embodies the same basic features of the single-channel Viso Cardiette It contains four separate channels which give four simultaneous tracings There are three types of input circuits and a wide, controllable range of sensitivities and paper speeds Each of the four channels incorporates four push pull stages of amplification with the last three stages direct-coupled The first stage (ECG preamplifier) is capacity-coupled to the last three stages A balanced at tenuator or double network of resistors is placed between the first and second stages It is designed in order to reduce large signals by definite ratios to amplitudes suitable for registration without overloading the DC amplifiers It has five steps of attenuation permitting the recording of signals 4 20 100 400 and 1000 times the average normal amplitude A sixth position of the attenuator ($\times 1$) feeds the signal to the amplifier without reduction of voltage The over all sensitivity of each chan nel is such that it gives 1 cm deflection for 1 mv of AC signal directly into the grids of the first stage push pull tubes On the other hand 50 mv are required for an equal deflection when the signal is applied to the DC amplifier input jacks Because of the design of the amplifier, balanced circuits can be fed directly to the first or second stage grids in push pull by grounding one of the two grids of the first or second stage an unbalanced (single-ended) circuit can feed the ungrounded grid

The sensitivity of the ECG and DC recording systems for each position of the attenuator is shown in Table 29

The conventional electrocardiographic leads are recorded through the ECG amplifier (1,2 3 aVR aVL aVF V,CR CL CF) Sphygmograms phlebograms and other low frequency tracings (apex cardiogram regional cardiograms epigastric

tracing) can be recorded through the ECG Pulse input jack A phonocardiogram can also be recorded for timing even though the frequency of the system does not allow a perfect reproduction of the rapid sound vibrations Signals containing slowly varying or constant components like intracardiac or arterial pressures respiration etc are recorded through the DC amplifier

TABLE 29 ATTENUATOR STEPS POLY VISO CARDIETTE

Attenuator setting	*Sensitivity for 1 cm deflection (volts)* Through ECG amplifier	Through DC amplifier alone
×1	0 001	0 05
×4	0 004	0 20
×20	0 02	1 0
×100	—	5 0
×400	—	20 0
×1000	—	50 0

The constants for each galvanometer are as follows

Sensitivity	10 ma /cm
Torque developed by 10 ma in coil	200 000 dyne/cm
Undamped natural frequency	45 cycles/sec
Maximum undistorted deflection	2 5 cm each way (from center)

Under critically damped conditions 80 per cent of a final deflection is reached in 0 01 second

The paper speeds selected through a gearshift are 50 25 10 2 5 1 0 0 5 and 0 25 mm per second The speed of 25 mm per second is the conventional speed for clinical electrocardiograms Special amplifiers arranged to replace one or more of the DC amplifiers can be added Then the Poly Viso can be adapted to record ing of other electrophysic and biologic phenomena (stresses strains temperature or pressure variations) An *instomatic* device is provided to protect the writing arms when a selector switch is moved from one position to another An automatic limit circuit also protects the writing arms against too marked deflections

Cardioscopes

These instruments are designed for clinical demonstration on a slowly moving screen of electrocardiograms vectorcardiograms and other phenomena They can be connected with a normal electrocardiograph having the proper input jack (San born Stetho Cardiette Sanborn Poly Viso Cardiette) or constitute a separate assembly

The *Cambridge Electronic Cardioscope* consists of (1) an amplifying stetho scope unit with multiple stethophones (2) two electrocardiograph amplifiers with multiple lead selector switches and (3) a large cathode ray tube having a long per sistence screen The following phenomena may be viewed the electrocardiogram in any lead the vectorcardiogram of several lead combinations the heart sounds

and murmurs (also heard by audiophones) A recorder and reproducer is available in order to record interesting or uncommon phenomena

PHONOCARDIOGRAPHS OR STETHOGRAPHS

The heart sounds can be amplified (amplifying stethoscope) and recorded (stethograph) by a special equipment which consists of (1) a microphone which picks up the rapid acoustic vibrations of the chest wall, (2) an amplifier which serves to intensify the small output of the microphone, and (3) an oscillograph which records the tracing on a film Since the tracing of the heart sounds is some times of difficult interpretation, it should be recorded simultaneously with an elec trocardiogram a cardiogram or a phlebogram, which are used for 'timing the various waves

In *Einthoven's Phonocardiograph* the heart sounds picked up by a common stethoscope were directed to a carbon granule microphone with an interposed adjustable leak valve The electrical pulsations originating into the microphone were adjusted in intensity by a rheostat, passed through a transformer and re corded by the metal-coated quartz string of a galvanometer The string was tight ened in order to record high pitched vibrations Several defects of this device (noises originating in the leak valve and in the undamped carbon particles of the microphone distortion of the sounds by the transformer), reduced the practical value of the apparatus

Several subsequent modifications although notable did not permit an accurate transcription of the heart sounds until the *piezoelectric crystal microphone* was perfected In this device the output potentials are proportional to the stresses in duced by the acoustic waves These varying potentials pass into a vacuum tube amplifier similar to those used in the amplifying electrocardiograph The amount of amplification is regulated by a volume control device Audiophones for ausculta tory control of the degree of amplification, and a D Arsonval type of galvanometer capable of responding to the frequency bands of the sounds are connected to the output of the amplifier As in the amplifying electrocardiograph the galvanometer coil carries a mirror and oscillates in response to the electrical pulsations A beam of light is reflected by the mirror upon a moving bromide film The combination of the longitudinal motion of the paper with the transverse movement of the light beam produces a graph of the sounds plotted against time

In crystal microphones Rochelle salt crystals are commonly used (sodium po tassium tartrate) because the piezoelectric constant of this salt is more than one thousand times that of quartz or tourmaline The fundamental frequency or natural period, of a bimorph crystal is approximately 10 000 cycles/sec When a diaphragm is coupled to the crystal the fundamental frequency of the combination is lowered to a few thousand cycles per sec This is still well above the upper frequency limit encountered in phonocardiography The microphone case incorporates an acoustic high pass filter which eliminates the low frequency vibrations of the chest wall having no acoustic significance and protects the crystal from sudden stresses such as those produced by applying the microphone to the chest The same system can be used in making a microphone with a flat frequency response from near zero to

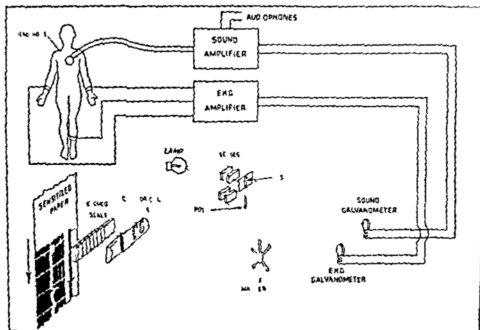


FIG 269 Sketch of an apparatus for simultaneous recording of electrocardiogram and phonocardiogram (From Rappaport and Sprague courtesy of C V Mosby Co)

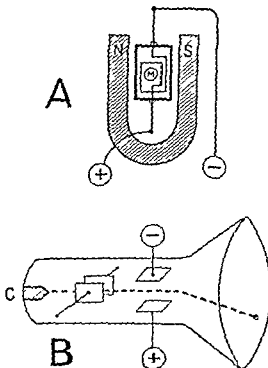


FIG 270 A Sketch of an oscillograph *B* sketch of a cathode ray tube (From *Fattorusso*)

several hundred cycles per second This microphone called the *linear microphone* is connected by a rubber tubing to a cup and can be used for electrical recording of the arterial or venous pulse or other mechanical phenomena, overcoming the imperfections of the mechanical and optical capsule types of sphygmographs

The above mentioned principles were used in the design of the *Sanborn Stetho Cardiette* which includes two independent channels of different electrical characteristics one for amplifying sounds and the other for electrocardiography The galvanometer employed in the electrocardiograph is a D Arsonval type moving coil carrying a mirror it has a deflection time of approximately 0 01 second The amplifier system has no logarithmic decay for more than 0 2 second, a gradual one thereafter Therefore this channel can be used also for recording other physiologic phenomena *in connection with either a piezoelectric sphygmographic attachment* (linear microphone) or an electrokymograph The stethographic channel can be used only for the recording of heart sounds The tracing appears in the upper strip of a 6 cm high bromide film The camera should be run at 25 mm /sec for recording the ecg and at 75 mm /sec for sounds

Two types of crystal microphones are provided for sound tracings The first is able to pick up all the vibrations of the chest wall, except the very low pitched having no acoustic value (0–10 cycles/second) it is called the *stethoscopic microphone* The other has an acoustic filter excluding the low pitched vibrations for which the human ear has a poor sensitivity, the resulting tracing is a picture of the sounds as they are heard by the human ear it is called the *logarithmic microphone*

An attachment is provided for one or more audiophones The *audiophone* is an electromagnetic telephone receiver, which converts the amplified electrical pulsations into equivalent sounds similar to those normally heard with a stethoscope In this way the observer can control the correct recording of the sounds Another attachment can be used for the *cardioscope* a useful complement in clinical demonstrations The electric power of the Stetho Cardiette is supplied by batteries

This two-channel model has been improved in the *Sanborn Twin Beam Cardiette* This consists of (1) a recorder and output amplifier unit, with controls and (2) two removable and interchangeable amplifiers which provide for simultaneous or separate registration of electrocardiogram (or slowly varying inputs) and phonocardiogram It is a portable apparatus operated by AC The power supply is electronically regulated to compensate for shifts in line voltage from 105 to 130 volts without materially affecting the baseline The two-channel output amplifier operates two identical galvanometers which have undamped natural frequency of 500 cycles/second but are damped to about 71 per cent of critical damping corresponding to a deflection time of one millisecond

The ecg DC amplifier is a separate plug in unit and consists of a preamplifier stage and a driver stage The preamplifier stage is based on a condenser coupled to the driver stage which is in turn directly coupled to the output stage The entire amplifier is a push pull unit It provides for amplification and registration (through the recorder unit already described) of electrocardiograms, pulse type signals slowly varying signals and constant (or DC) signals The normal over all sensitivity of the ecg amplifier is 0 5 mv /cm The normal sensitivity of the DC

amplifier is 20 mv /cm The input impedance of the DC amplifier is 5 0 megohms grid to ground or 10 0 megohms grid to grid Direct access to a preamplifier grid is available as stated below through the pulse input jack and the input impedance is exceptionally high There is a six step attenuator having ranges from 1000 1 to 1 1 plus a calibrating position and an *OFF* position

The phono amplifier contains a two stage amplifier between the microphone and the output amplifier of the oscillograph unit Audiophone output is made available through a separate output stage The input impedance of this unit is 30 ohms and the amplifier is equalized so that an over all frequency range of 1000 cycles/second is achieved The normal sensitivity of the amplifier in the frequency range near 100 cycles is such that a deflection of 1 cm will be obtained for a sound pressure of 0 16 dynes per square cm (58 decibels above the standard reference threshold of 0 0002 dynes per square cm) Upon auscultation by audiophone the reproduction of heart sounds closely duplicates the phenomena heard through a conventional stethoscope A switch permits the choice of either a logarithmic or a stethoscopic type of record without changing the characteristics for auscultation which is always logarithmic on account of the characteristics of the ear Both the microphone and the audiophone are impervious to moisture and not affected by temperature changes

The Twin Beam Cardiette may be equipped originally with two ecg DC amplifiers or with one ecg DC amplifier and one phono amplifier or with two phono amplifiers Complete interchangeability of these amplifiers permits the operator to set up any combination at any time

AC or DC-operated string electrocardiographs were also modified by the addition of a stethographic unit consisting of a crystal microphone a vacuum tube amplifier and a moving coil D Arsonval type galvanometer (*Cambridge All Electric Electrocardiograph-Stethograph* and *Simpli Trol portable Electrocardiograph Stethograph*) The sound tracing is recorded on the upper part of a 6 cm high photographic film The same principle is applied for the *Mobile Type* and the small portable *Simpli Trol* A pulse recording attachment is also provided which permits the mechanical recording of the arterial or venous pulse simultaneously with any tracing recorded by the electrocardiograph It consists of a recording tambour a receiver for application to the artery or vein and the connecting tubing The recording tambour which is contained in a small metal housing mounted on top of the projection lens tube consists of a diaphragm to which is attached a light lever of metal tubing which projects downward into the lens tube so that its image is projected into the camera in the same manner as the shadow of the galvanometer string Consequently the movements of this arm are magnified by the ocular lens Two types of receiver are furnished For recording the radial pulse a modification of Lewis glycerine pelotte is used For recording the carotid or jugular pulse a light molded plastic cup is provided which adheres by suction to the skin over the artery or vein

BALLISTOCARDIOGRAPHS

Mechanical Methods

In Starr s original *spring damped bed,* the movement of the bed suspended from the ceiling was magnified by a moving mirror and a light beam system and recorded by a moving film camera Ballistocardiograms can be obtained also by means of a *mechanical pressure recorder* (so called sphygmographic method) A receiving membrane is connected by a rubber tubing having a leak to a recording membrane, as in a pulse recorder This is applied to the vertex of the patient s head

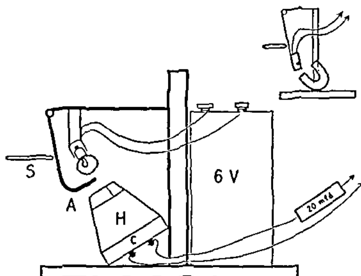


Fig 271 Sketch of Dock s magnetic ballistocardiograph (insert) and of a photoelectric ballistocardiograph (from Dock and Taubman courtesy of the *Am J Med*)

Electromagnetic Method

This simple method records the ballistocardiogram by detecting the velocity of bodily motion more than its magnitude A coil of fine copper wire, supported by a crosspiece placed across the shins of the patient moves within the field of a permanent magnet the resulting electrical current is transcribed by a galvanometer attached to the coil Many arrangements are possible the most used include (a) a 1 ×⅜ ′ coil of 8000 turns of No 40 wire wound on a bobbin (b) a HS2V Alnico horseshoe magnet

Photoelectric Method

Many devices can be used A beam of light can be moved by the shaking body across the window of a phototube or an occulting strip illuminated by a standing light, can project its shadow on the same window

The first principle is used in the *Sanborn apparatus* which consists of two units The first is a crosspiece placed over the shins of the patient it contains a constant

light source (supplied by a 3 volt battery) an optical system a voltmeter and a filament temperature control The second consists of an adjustable pedestal placed on the table next to the patient and housing a phototube As the beam of light from the frame swings rhythmically across the rectangular hood of the phototube a gal vanometer (a common electrocardiograph connected by the lead cables to the photocell unit) traces the ballistocardiogram The sensitivity of the recording elec trocardiograph is set at 1 cm /mv

A switch is installed at the base of the photocell unit below the binding posts for the electrocardiograph cables With the switch at *IN* a filter is included and the characteristic of the entire unit approximate those of Starr s ballistocardiograph The wandering of the baseline due to respiration is reduced a deeper K wave is registered and other minor modifications take place With the filter switch at *OUT* the baseline wanders with respiration This can be obviated if the tracing is recorded during apnea

When taking a ballistocardiogram the electrocardiograph lead selector should be turned to Lead 1 (except in the Poly Viso or Twin Beam) The beam spreading from the light source must cover *half* of the viewing scale of the phototube A gentle caudad push applied to the patient s soles should cause an upright deflection of the tracing Otherwise the photocell unit should be so moved that the light field covers the opposite half of the viewing scale with the edge of light meeting the hairline

ELECTROMANOMETERS

The *Sanborn electromanometer* is an instrument for direct pressure measurements (intracardiac intra arterial intravenous cerebrospinal or other) through the use of catheters or needles or for measurements of pneumatic pressures The assembly of the instrument transforms pressures into equivalent electric potentials these are amplified to values sufficient for galvanometers having a sensitivity of not less than 0 1 ma /cm The instrument can be operated with any electrocardiograph in the case of the Viso Cardiette an included amplifier intensifies the electric output of the electromanometer

The apparatus consists of two units The main unit includes a bridge circuit with electrical control a manometer and a transducer with its control valves The trans ducer contains a condenser microphone which acts as the pressure sensitive element of the AC bridge circuit this is an electric network with input and output terminals The circuit and the components are so arranged that a balance condition can be obtained when no output voltage appears even though an input voltage is applied If however one of the elements of the bridge circuit is varied the balance condi tion is destroyed and some fraction of the input voltage appears at the output terminals The magnitude of this output voltage depends upon the degree of unbal ance and is directly proportional to the magnitude of change occurring in the variable bridge element When the condenser transducer is stimulated by pressure the bridge becomes unbalanced and the unbalanced voltage is amplified rectified and fed to the electrocardiograph (or other recorder)

The hydraulic system contains two valves, one connects the bottle to the trans ducer (BT) or to the catheter (BC), or the catheter to the transducer (CT) the other valve connects the water manometer to the transducer in order to record low pressures

The second unit consists of the power supply box which is used in order to select the sensitivity range and the *registration of either the mean pressure or the instan* taneous pressure

In general, the electromanometer makes use of a crystal microphone of medium sensitivity For special studies a high sensitivity microphone can be used, then

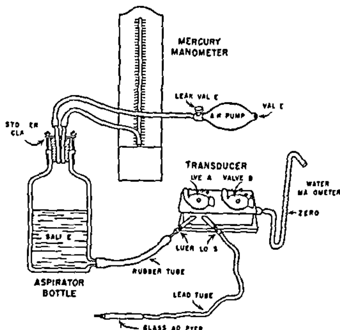


FIG 272 Hydraulic system in the Sanborn electro manometer

small changes in pressure like those caused by low frequency vibrations of the chest or respiratory movements can be recorded

A switch permits recording either mean pressure or the pulsations of a variable pressure

ELECTROKYMOGRAPHS

The first practical apparatus was built by the Cambridge Co Several modifica tions tending to an increase of the magnitude of amplification without proportional increase of the artifacts were embodied in another model built by the Sanborn Co In both apparatus the main part is an electro multiplier phototube (RCA 931 A) which consists of a light sensitive photocathode a system of nine secondary emis sion electrodes (dynodes) and a collector anode The phototube has a maximal

response to light of 4200 Å which is in the blue region The sensitivity is abou 70 per cent of the maximum for green radiation

When the photocathode is exposed to light a proportionate number of electrons is released and immediately attracted to a dynode which is at positive potentia with respect to the photocathode The surface of the dynode is treated for second ary emission so that each electron which originates at the photocathode displaces several additional electrons at the first dynode These secondary electrons are then directed to a second dynode which is at a potential more positive than the first dynode surface and they in turn displace more electrons Since this multiplication process is cumulative in the nine stages of amplification the maximum over all current of amplification attainable is approximately one million times

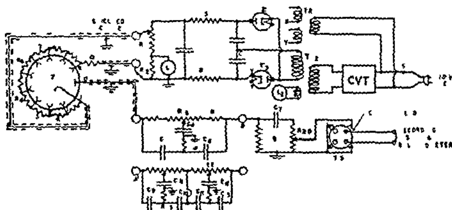


Fig 273 Scheme of the Cambridge electrokymograph (from Henny and Boone courtesy of Charles C Thomas)

The author has used subsequently an RCA 1P21 phototube This is similar to the 931 A but is capable of an amplification of about two million times if the tube is operated at 100 volts per stage Artifacts are minimized by the use of this tube It should be kept in mind that the high voltages at which this tube is operated are dangerous and that shielding should be perfect

A strip of Patterson B screen is cemented to the glass envelope of the multiplier phototube directly in front of the photocathode When the x rays strike the fluor escent screen the light emitted by the screen is picked up by the photocathode transformed into equivalent photoelectrons and amplified by electron multiplica tion Thus the electric output of the phototube varies in proportion to the move ment of the silhouette within the opening of the slit The output of the tube is in turn fed into the electrocardiographic channel through a potentiometer which acts like a sensitivity control

Three forms of interference should be eliminated electrostatic radiation flicker due to the cyclic discontinuity of the x ray emanation and fluctuations of the power line

The first is easily eliminated by complete shielding of the electrokymographic circuit (the recording electrocardiograph is sufficiently shielded) To suppress the flicker interference a tuned filter attenuating the flicker interference approximately one thousand times was used in the Cambridge model With the greater amplification obtained in the Sanborn model, this attenuation was insufficient, therefore a two stage resistance capacitance type parallel T network attenuator was used this attenuates the flicker at least one hundred thousand times Moreover, the tuned filter reduces the effective deflection speed of the galvanometer (from 0 01 to 0 02 second at least) which is less affected by the resistance capacitance attenuator

A further modification, described by Swedish workers, was based on the use of a DC amplifier, replacing the resistance capacity filter with an inductance capacity filter in order to obtain correct timing

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